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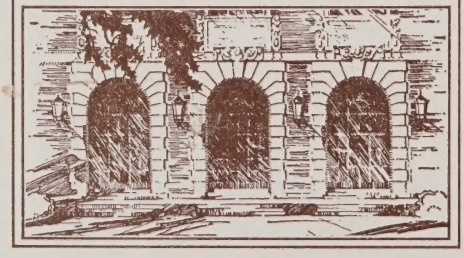

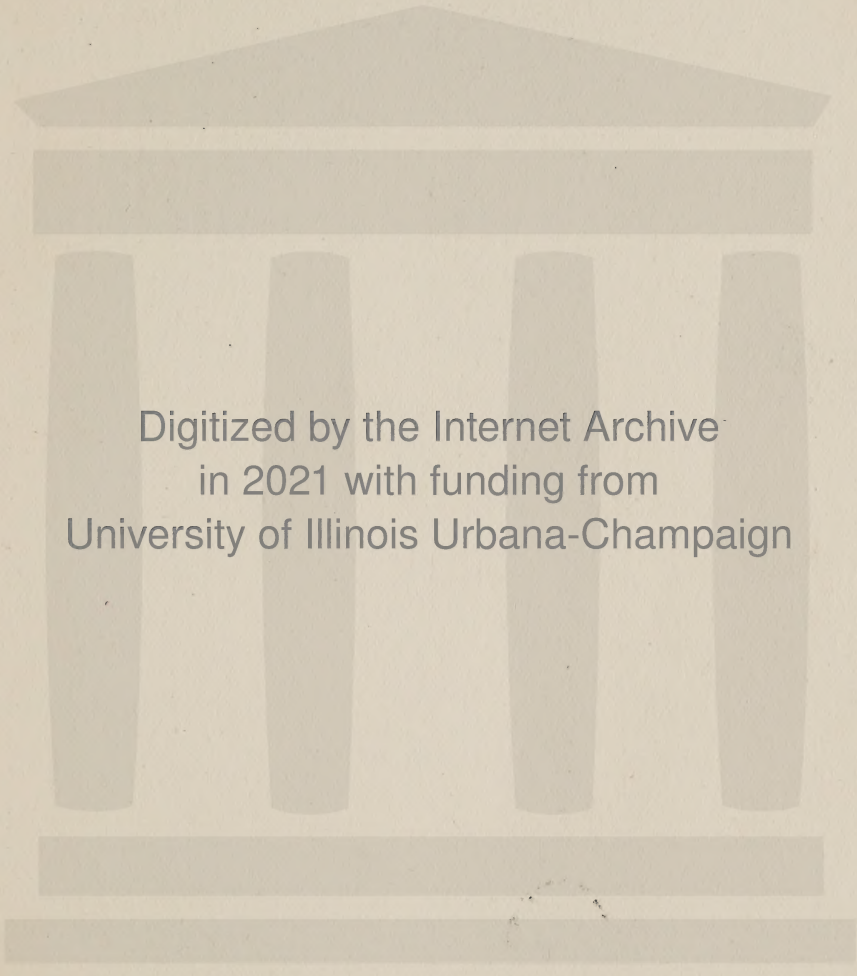
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PRESIDENTIAL ADDRESS.*

By Joseph Collins, M.D.

In opening this, the Twenty-eighth Annual Meeting of the American Neurological Association, it may be permitted your President to dwell for a few moments upon the prosperity of our Guild. A prosperity that is shown by its full membership, its waiting list, and by the character and the amount of work that it has done, particularly in the last few years. Finally, I desire to say a word on the influence that this Association has had in promoting mutual respect and good fellowship among those laboring in the field of neurology throughout this country.

When one examines the transactions of the Association in its earlier years, he sees at once the great change that has come about. In 1877 there were 36 members in the Association, and at the annual meeting held at New York the attendance was 12. Only one member came from out of town, and he came, probably, because he was President. Six years later, in 1883, the membership had increased somewhat, but only 14 members attended the meeting in that year. About this time many of those who have since been the vertebræ and brain of the Society, came in, and by their suavity and

*Read before the American Neurological Association, June 5, 1902.

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earnestness counteracted the internal dissension which threatened to wreck the Association. At the same time they gave it a scientific atmosphere which has succeeded in bringing the Association to its present proud place among the national societies.

Today, the membership is full, there are several names already familiar to neurologists on the waiting list, and the number of papers presented is so great that they cannot be got through with in the three days which we devote to our meeting. As to the quality of the work indicated by the program, all that can be said is that it is our best. We should not be expected to put further estimation upon it.

In a large body such as this, Death, the Inevitable, takes yearly from our ranks. At this meeting the Chair fortunately has but two to record. The brightest light in neurology on the Western horizon went out in the death of Dr. Eskridge. A Pennsylvania man, driven from a career on his native heath by tuberculosis, who obtained within a few years in the land of his adoption a secure place in the affections of his fellow workers at Kansas City, and in the estimation of his fellow specialists in the entire country. As a clinical neurologist, Dr. J. M. Eskridge had few superiors. As an earnest, conscientious, hard worker, he had none.

Dr. Charles Henry Brown, who was well known to all of us, found the *Journal of Nervous and Mental Disease* in a defunct state. After several years of struggle, during which time he gave to it his best energy and his most strenuous concentration, he had the satisfaction of knowing that it stood the test of comparison with the best of the special journals.

A pioneer in neurology died a few weeks ago. When Meredith Clymer began his studies of nervous disease the field was as trackless and unbroken as the region beyond the Alleghenies when Daniel Boone broke its virginal soil.

Two generations have come, and are going since then, and while the opportunity offers, one of the latter generation desires to record an appreciation of the truly remarkable work which this Nestor of American neurologists did nearly a half century ago. He had withdrawn from the field before this Society

was organized, therefore his name is not associated with its annals. But in the proceedings of that great unorganized society, the "World's Best Work," his name is distinctly to be read.

I venture to believe that the President of this Association can serve its members in no more satisfactory way than by putting before it briefly as the time compels, a succinct epitome of the most important work that has been done in neurology during the term of his office. This I shall now attempt to do. Although no epoch-making contributions have been recorded, examination of the literature shows that there has been no dearth of effort to solve the many problems in anatomy and pathology with which we are confronted.

The first and most important question is: What is the status of the neurone doctrine today? Is the neurone an anatomical, pathological, developmental and functional unity, such as the vast majority of neurologists have believed it to be during the past ten years, or is it necessary in view of recent investigations to change our conception of the neurone, not only in regard to it as a unity, but as to its mode of communication or connection with other neurones.

Five years ago many believed that Apáthy dealt the neurone theory a staggering blow when he showed that in the ganglia of the leech and the earthworm there was a fibrillar network which passed without breech of continuity between and through the bodies of nerve cells. Although Apáthy's claim has not been verified by others, or further substantiated by himself, the investigations of Held tend to corroborate, in a measure, Apáthy's claim. Held has shown that in the cells of several nuclei of the pons, in the cortex of the cerebellum, and in the anterior horns of the cord of the rabbit, it is possible to trace an increasing intimacy of union between terminals of axones and cell-bodies. About the time of the animal's birth the terminals come in contact with the cell-bodies and gradually fuse with it by a process which Held calls "concrescence." This concrescence is first evidenced by a layer which is more highly refractive than its surroundings, and, later, by slightly different texture and staining reaction. There have not been lacking other

investigators who have maintained with more or less convincing argument that there is structural connection between neurones. Hill[1], as you know, described a continuity by means of fine, cord-like threads, stretching across from fiber to fiber, and Verworn[2] says that in adults in many cells there is direct continuity.

The results of these investigations are not in reality antipathic to the neurone theory at all, when the theory of the neurone's individuality and discontinuity is not held too strictly,* nor do all of the investigators just mentioned believe that their findings invalidate the neurone doctrine. Held, for instance, distinctly disclaims such imputation. The concrescence of Held does not bespeak a structural communication between the neurones. It assumes that embryologically the units are anatomically independent, but that in later development there is a fusion without structual blending. It seems to me that it must be conceded that ontogenetically neurones become more intimately united in series, and that such intimacy is structurally heightened by use.

Although the conclusions of the authors just mentioned can be reconciled with the neurone doctrine, as it is interpreted today, the recent work of Bethe[3], and Ballance and Stewart[4], presents greater difficulties. Acceptation of their conclusions seems to demand that the position which neurologists have taken in reference to the unity of the neurone be materially modified. These authors, attempting to solve the problems connected with the healing of nerves, seem to have demonstrated that the process of regeneration of peripheral nerves is conditioned by protoplasmic proliferation of the sheath of Schwann, the process being one of karyokinesis from pre-existing cells; and that it is not at all influenced by the ganglion cells from which it has been severed. Bethe, working with puppies and rabbits, has shown that after a peripheral nerve is completely cut across and the distal end wholly degenerated, complete regeneration of the distal or peripheral segment occurs; not only anatomical regenera-

*As Verworn truthfully says, "Contact, anastomoses through fibrillæ or concrescences do not change the neurone theory any more than the intercellular bridges change the cell theory."

tion, but physiological as well, which is, however, more complete in the lower animals. Ballance and Stewart, in their work on the Healing of Nerves, corroborate Bethe in nearly every detail. Bethe has shown further that if the peripheral portion of a nerve which has regenerated, and which has been kept from uniting with the trunk from which it was originally severed, be cut across again, the central end, that is the end which has now no axonal continuity in either direction, does not degenerate. This shows that not only the new axis cylinders develop from preëxisting cells and are in no way outgrowths from axis cylinders in connection with cell-bodies, but it reduces the matter to an admission that a portion of nerve cut off from all connection with the ganglion cell, can, under certain circumstances, take care of itself. This is so revolutionary that we must have very substantial corroboration of it before it can be accepted.

Bethe further maintains that in puppies, at a time when as yet no nerve fibers have appeared in the spinal cord, the locality which the fibers occupy later is indicated by rows of cells which can be followed from the cord to the primitive muscle substance. From this he infers that the first *anlage* of the nerves consists of cells. This multicellular development of the axis cylinder is, as is well known, the claim of Balfour, Beard, Kupfer, Dohrn, Tizzoni, and others. It is entirely irreconcilable with the embryological results of His and of Köl-liker, not to mention others of the great number who teach that each axone is the mere outgrowth of a process of a nerve cell. It is no less directly contradictory to the teachings of Huber, Stroede, Vanlair, Harrison and others, that regeneration of the peripheral nerves after division of them is by a process of downgrowth from the axones of the central segment.

In further corroboration of his contention regarding the development of the axis cylinder of the neuraxone, Bethe maintains that when the first fibers are to be seen coming out from the spinal cord one may find contemporaneously primitive nerve fibers in equal or in greater number in the muscle, which speaks in favor of the view that the primitive nerve fi-

bers develop along the whole line at about the same time. With the same distinctness that prolongations of the neuroblasts from the motor roots are seen, one can see remote from the spinal cord the central prolongations of those bipolar cells of the primary *anlage* of the nerves which stream far distant into the cord, and from such pictures he concludes that the peripheral "nerve cells" develop the nerve fibers as far as the ganglion cells. In the main, the experiments of Ballance and Stewart corroborate those of Bethe, particularly in the conviction that the true source of the regenerative process in peripheral nerves is not the cells of the anterior cornua or posterior root ganglion, but the neurilemma cells of the nerve trunk itself. Upon the question of the embryonic origin of the neurilemma cells they are not decided. They believe, however, that the peripheral nervous system is to be regarded as composed of chains of neuroblasts, fused together to form continuous axones enclosed within medullary and neurilemma sheaths. For them the presence of a neurilemma sheath is of fundamental significance, since upon the presence of neurilemma cells depends the possibility of regeneration.

For these investigators the neurone theory is no longer tenable. Bethe has also convinced himself that the theory as accepted at present by the majority must be foresworn. He has suggested that the entire nervous system be conceived of as made up of a large number of cell-societies which are brought into functional interrelationship by means of the neurofibrillæ. One may call such a cell-society a neurone, a ganglion cell being its morphological center of gravity but not its trophic and functional center. A cell-society may be made up of few or of many cells. In addition there would be other cell societies which lack these morphological centers, for instance, the intracentral fibers, the existence of which Bethe seems to be as certain of as is Nissl. Other cell-societies (muscle, gland, reception cells, etc.) are in inner functional and trophic relationship with the nerve cell-societies, this relationship being founded on the fact that the en-

tire animal is an organism, but not the cells which constitute it.

This conception of Bethe seems to me very rational. If it can be maintained that the neurone cell-unit is made up of several cells it will prove a valuable hypothesis. To discuss it, however, would presuppose an acceptation of Nissl's claim that the larger part of the human gray matter is made up of Apáthy's fibrils in a free condition outside of cells, or, as he calls it, a specific nervous substance derived probably from the protoplasm of the nerve cells, which represents the highest stage of differentiation of the cell plasma within the animal body. It does not seem to me that the time is yet ready to pass judgment upon this claim, because the principal evidence for the existence of this substance is theoretical, being based largely on the assumption that the higher the grade of development of an animal the fewer nerve cells inhabit an equally large volume of the cortex, and that in the human cortex it is impossible to account for the whole mass of the gray matter as nerve cells, their processes and neuroglia; for when these are subtracted there remains a very considerable amount of substance not accounted for.

The work of Bethe has only been published in abstract, and final judgment of it must therefore be reserved. The work of Ballance and Stewart, however, has been published in detail, fully and beautifully illustrated. As their conclusions are in accord with a small number of thoroughly accredited investigators, they must be accepted, until such a time, at least, as they are shown to be erroneous by other investigators working with the same method. It is possible that we shall have to modify materially some of the teachings of the neurone doctrine, but so far, I venture to believe, nothing has been discovered with which the neurone doctrine properly conceived cannot be made to harmonize.

In reviewing the field of clinical and pathological neurology I shall confine myself very briefly to some of the most important work. American contributions will not be considered; not because there are not many milestones indicating the progress of neurology in this literature, but because the Chair

does not allow itself the liberty to discuss *ex cathedra* your work.

The most important contribution to the subject of localization of function in the cerebral cortex is the report on the physiology of the cortex of some of the higher apes, by Sherrington and Grunbaum[5]. Their investigations on chimpanzees, orangs and gorillas extend and correct our knowledge of localization. In the main they corroborate the investigations of Bevor and Horsley. In two important particulars, however, they differ; that is, first, that stimulation of the ascending parietal gyrus causes no movement; and second the representation of the movements of the chest and abdomen between those of the hip and shoulder, and those of the neck between those of the face and thumb. Flechsig's[6] last contribution to the myelogenetic localization of the cerebral cortex, contains no important facts that have not before been set forth. Formerly he distinguished forty myelogenetic areas. This is now reduced to thirty-six. Every area possesses a special anatomical position and therefore a special functional importance. He maintains that the inferior longitudinal fascicle is an important projection system (the real optic radiation) and that the cingulum is likewise a projection system. These have recently been described by Monakow and Dejerine as association systems.

Munk[7], in a third communication on the extent of the sensory sphere in the cerebral cortex, concludes from critical sifting of the experimental, clinical and pathological material, that neither the cortex of the frontal lobes nor the cortex of the parietal lobes have special localization of the higher psychical functions. Flechsig's teachings relative to the association areas are erroneous he maintains, and there is no special portion of the brain that is uniquely concerned with the psychical functions. In this connection it is interesting to refer to the conclusions of an American surgeon, Charles Phelps[8], who maintains from a study of brain injury and disease that the left prefrontal lobe is the important seat of the mental faculties.

During the past year there has been much discussion of the reflexes and tendon-jerks. A few new reflexes like the supra-orbital, palmar, scapular and a number of pupillary reflexes have been described. Some of these are awaiting corroboration as to their existence and elicitation, while the final value of others is still to be determined. By far the most important work in this direction, however, has been the corroboration of the statement made by its discoverer that the big toe phenomenon (the so-called Babinski reflex) is a semiological indication of no less importance than the knee-jerk or the ankle-jerk, and that when it is present in typical or genuine fashion, that is, a slow dorsi-flexion of the great toe, depending upon vermicular contraction of the muscles, it is absolutely pathognomic of degeneration in the cross pyramidal tracts. The existence of the phenomenon in children before they have learned to walk has been of aid in putting interpretation of the physiological basis of the reflexes and in determining the function of the pyramidal tract, which latter point is still awaiting a final and satisfactory conclusion.

Of some practical, but of much more theoretical importance, is the "tibial phenomenon" to which attention has again been called by Strümpell[9], he having described it first a number of years ago. It consists of dorsi-flexion of the foot, occurring involuntarily and beyond the patient's control when the thigh of a hemiplegic limb is flexed upon the abdomen. When the normal thigh is flexed upon the abdomen, the foot of that side falls into plantar flexion, and continues to droop as the thigh is flexed. In a hemiplegic extremity, or in a leg motorily incapacitated by lesion of a pyramidal tract, the reverse of this takes place. Strümpell points out that the area of the pyramidal tracts in the lower dorsal and lumbar region is not commensurate with the immense amount of peripheral neuraxon matter which goes to these extremities, and infers that the subject of the distribution of the central motor neurones in the cord may be a fruitful subject for study.

An important contribution to the topography of the spinal cord and the oblongata is that of Thiele and Horsley[10], who trace the degenerations in the central nervous system in a

case of fracture dislocation of the spine. They conclude that the fibers of the direct cerebellar tract terminate mostly in the superior vermis; that Gowers' tract is, as Mott has shown, a complex of several systems, spino-cerebellar, spino-quadrigeminal, and spino-thalamic. Other questions of importance relative to the fasciculus spino-quadrigeminalis and the fasciculus spino-thalamicus, whose solution has been facilitated by this work can here only be hinted at.

In the domain of pathology comparatively little work of any great importance has been done. One of our own members[11] has published in Brain certain changes in the central nervous system which it is maintained constitute parenchymatous systemic degeneration. The change is that of axonal reaction in the cells of Betz and decay of the myelin sheath of some of the corresponding sensory fibers. These cases clinically do not permit of classification, but the alteration has been found to occur in peculiar forms or end stages of depressive disorders, near or after the climacteric, alcoholic, senile and phthisical idiocy, and perhaps also general paralysis. I am pleased to note that Dr. Meyer will demonstrate some specimens of this condition at this meeting.

It has been quite generally believed that many or all nervous diseases now classified as functional will eventually resolve themselves into conditions that are discernible to mechanical and chemical technic. Although this will never be realized there can be no question that many of the diseases which are now classified as functional or dynamic and the result of intoxications or infections, will eventually be so interpreted. Gastric tetany is a condition which is theoretically supposed to be the result of the activity of a toxin generated in the stomach upon the peripheral neuro-muscular apparatus, and Rossolimo[12] has recently found in a case of gastric tetany changes in muscles and nerves characteristic of mild inflammation. The inference is that in the milder cases an autointoxication results which is comparatively transient, but in the severer cases extensive lesions which are protracted in their duration may be the result.

From the time of the publication of the researches of

Achalme[13], Riva[14], Apert and Triboulet[15], Poynton and Paine[16], showing that rheumatism is an infectious disease depending upon a specific organism, those who believe in the close relationship between chorea and rheumatism have been on the lookout for material that would show the existence in acute chorea of organisms similar to those described by the investigators mentioned above. Preobajensky[17] has recently published a case in which there was found at the autopsy the lesions of hemorrhagic lepto- and pachymeningitis and acute infectious cerebritis all of streptococcus origin. Cultures of streptococci were obtained from the blood and from the brain. The writer remarks that the necessity of differentiating the different forms of infectious chorea exists in order that a rational therapy may be applied. In such a case it would have been an antistreptococcus serum. But we will have to make long strides in hematology before such differentiation can be made.

The pathological anatomy of general paresis is by no means so thoroughly understood as it is commonly believed to be. Some important studies recently made in this disease are those of Shaffer[18], of Budapest, and Storch[19], of Breslau. The former has shown that the disease has an elective affinity for Flechsig's association centers, that is the frontal, parietal, post-central, insular, the second and third temporal gyri, and the gyrus fornicatus. He concludes that his findings support Flechsig's theories, and that the cortical degeneration of paresis is not an irregular diffuse process, but a regular localized elective or selective affection of the cortex. This is in the main corroborative of Storch, who has shown that in atypical cases of general paresis those portions of the brain were affected whose functional allotment is such that disease of them would be presupposed from a consideration of the principal symptoms.

A highly suggestive and intrinsically valuable contribution is by Mott and Halliburton[20] on the chemistry of nerve degeneration, in which it is shown that excess of choline, a product of the decomposition of lecithin, occurs in many nervous diseases, whose morbid anatomy consists in degenera-

tion of the nerve substance, such as general paresis and various diseases of the central and peripheral nervous systems.

A disease whose occurrence and causation is quite as mysterious as it was when first described, and the secret of whose existence is wholly concealed, is that known as family periodic paralysis. It is generally supposed by those who have worked at the subject that the disease is due to some form of toxemia. Westphal suggested this idea, and Goldflam elaborated it, and although different observers have suggested that the toxin acts upon different parts of the nervous system, they are all agreed upon the probability that the disease is due to a toxin. Unfortunately no light is thrown upon this subject by the most recent communication (Singer[21]). From a study of the electrical reactions in a patient with this disease, Oddo and Darcourt[22] conclude that the seat of the affection is the muscles, and they consider the disease a variety of myopathy, essentially functional in character.

Asthenic bulbar paralysis is another disease in which a toxic substance generated within the system is supposed to be the exciting cause of the attack. Although this subject has received a great deal of attention during the past year, and a few cases have been carefully studied post-mortem, nothing has so far been found that tends to strengthen this theory, or to substantiate the existence of any such poison. Of considerable interest, however, is the existence in the cases published by Laquer[23] and Goldflam[24] of malignant disease of the thymus in the case of Laquer, and of the lung in the case of Goldflam. In both instances there were changes in the muscles which were considered to be metastatic from the tumor. That such findings must be looked upon as coincident and not at all to do with the real pathogeny of the disease seems to me incontrovertible. The number of recoveries from this disease is by no means small, which would alone negative such an idea. I have had personally under observation during the past eight years a patient who has been free from any symptoms for more than three years.

It has always been a matter of great doubt with American neurologists that syphilitic polyneuritis exists. Cestan[25] has

recorded three cases which he believes establish the occurrence of this condition. Taken in conjunction with what has already been written on this subject by other European writers, it seems to be incumbent upon us to examine into the subject a little more closely.

Ferrier and Turner[26] have shown the relatively small functional value of the quadrigeminal bodies in man and in monkeys. Destruction of them causes only transitory symptoms. This is in entire harmony with the present trend of experimental physiology.

The hypophysis cerebri has been the field of much experimental work during the past fifteen years, and especially since its supposed relationship to the development of acromegaly. The conclusions of the different investigators up to the present time have seemed to be quite irreconcilable. During the past year a number of monographs and essays have been published by men whose reputation for reliable investigation has been already established. Casselli[27] states that complete abolition of the hypophysis produces in the first place a slowing of the respiration, an acceleration of the pulse, diminution of the psychical function, hypertonia of the muscles, convulsions, progressive cachexia, coma and death. In many respects the symptoms following extirpation of the hypophysis are similar to those of diabetes. The author concludes that this organ is necessary to the human economy, and that it has a specific internal secretion, a modification of which brings about grave alterations in metabolism.

The statements of Casselli are corroborated in great detail by v. Cyon[28], who, by the way, seems convinced of the etiological relationship between disturbance of the hypophysis and acromegaly. In contradiction to these investigators is the work of Lomonaco and Van Rymberk[29], who make not only a most critical review of the literature, but add the results of their own experiments. They conclude that the hypophysis is a rudimentary organ without general or special functional significance. They maintain that the symptoms which have been described as following extirpation of the

gland are due either to injury to structures adjacent to the pituitary, or the result of shock or infection.

Friedmann and Mass[30], who performed the operation of extirpation of the hypophysis in 18 cats, reached practically the same conclusion. The status of the function of the pituitary gland may, therefore, be said not to have materially changed during the past year.

The morbid anatomy of exophthalmic goiter remains as obscure as ever, although no end of research work and anatomical investigation is being done to solve the problem. Recently Kedzior and Zanietowski[31] have published a preliminary communication which sets forth some findings in a case of this disease of four years' duration. Death was due to an intercurrent attack of croupous pneumonia. In the nervous system the following changes were found: Fresh and ancient hemorrhages throughout the entire oblongata especially in the vicinity of the left olive, with distension of the blood vessels. The left restiform body was much smaller than the right. On microscopic examination the degeneration of this restiform body was evidenced by its waxy appearance in contradistinction with the deep red of its fellow of the opposite side. The authors incline to the belief that this change may be taken as the basis of the disease in this instance, though in just what way it caused the symptoms does not seem clear. The recoverability of exophthalmic goiter is the best argument that no such change as that described by these writers is responsible for more than its accidental occurrence. The hyperplasia of the thymus which was found by these writers has also been remarked by a number of others, including Dinkler[32].

The findings in the central nervous system of disease of many years' standing, such as paralysis agitans, are not usually looked upon as being of any value whatsoever indicative of the morbid anatomy of the disease. It is generally recognized that they may more legitimately be the result of the paralysis agitans than the cause. Therefore, the contribution of Walbaum[33] is of no service in interpreting the disease, a conclusion arrived at by the author.

The occurrence of the optic neuritis in lesions of the spinal cord has been studied by Taylor and Collier[34], and their conclusions may be regarded as a real contribution to semiology, corroborating as they do the observations of our own distinguished and lamented members, Seguin and Eskridge.

From an analysis of twelve cases, they conclude that optic neuritis of all degrees of severity may occur in connection with tumor, compression, myelitis, or hemorrhage affecting in some degree the upper part of the spinal cord.

The therapeutic value of electricity is a subject upon which neurologists differ quite as much in all probability as almost any subject that can easily be mentioned. The chief reason for this is that trustworthy data are not at hand to guide us in putting an estimate upon its value, and not all of us have the time and application to make such data for ourselves. What is needed is reliable experimental work such as that which gave electro-diagnostics a solid foundation. As an indication of work in the right direction I take this opportunity of mentioning an article by Jellinek[35] in which the changes in the tissues, principally the nervous system, produced by electricity, are set forth.

We may count that year barren in therapeutic suggestion that does not witness a new cure for exophthalmic goiter, but whether the prognosis of the affection is materially altered thereby is doubtful. Abadie and Collon[36] give their results with the intrathyroid injection of iodoform and ether, one part of the former to five of the latter. One cubic centimeter of this solution is injected into the thyroid gland. The method was introduced by Pitres, who claimed satisfactory results from it, and the investigators just quoted claim to have had equally gratifying experiences, claiming twelve cures out of twenty-four cases. It must, however, be said that the cure of a case of exophthalmic goiter has no particular significance. We must know what sort of a case it is, meaning thereby that some cases are cured by anything, while others withstand everything.

It is a matter of disappointment that the Roentgen rays have not been of particular service to the neurologist in his struggle with disease, either in their diagnosis or their cure.

But recently it has been shown by one of our own members, Mills, in conjunction with Pfahler[37], that it is possible that they may be of service in the recognition or corroborative recognition of some tumors of the brain.

Amongst the most important clinical contributions of the year is one from England.

Dr. Henry Head[38] a few years ago while still a hospital interne instituted and carried out an investigation on the relationship of disease of the internal organs to superficial pain radiating around the surface of the body, and the tenderness of its superficial covering, that put him at once into the first rank of neurologists and established his right to be heard attentively in any claim that he might put forth. In the Gulstonian Lectures for 1901 he has attempted to show how far the intrusion of stimuli arising from disorder of the viscera upon the nervous system are accompanied by changes in consciousness; in other words, the changes in consciousness associated with the reflected pains of visceral disease. His explanation of these changes in consciousness, that is, of hallucinations, moods, suspicions, and changes in memory and attention, is simple: Under normal circumstances visceral life takes place outside consciousness; the visceral field is pushed out of consciousness and its records remain only as latent dispositions. A complete change takes place when the reflected pain of visceral organs comes into existence. They crowd into consciousness, usurping the central field of attention. It brings in its train all those images and dispositions which exist normally at the fringe of, or entirely outside of, the field of consciousness. The barrier which the normal mind sets between conscious life and that of the viscera is broken down. The importance that this study has in the interpretation of such diseases as hysteria, neurasthenia, and some other of the so-called functional nervous diseases, as well as the relationship which exists between diseases of the viscera and certain mental disorders, including hypochondria, is so great that the subject should be pursued by every neurologist to whom the opportunity is offered.

From this brief and cursory reference to some of the

most important contributions of the year it is seen that there has been more than usual activity in the realm of neurology, and although no contribution that can be regarded as epoch-making has been made during that time, the charge that neurologists have become sterile and their fields of labor have gone to seed can scarcely be maintained. Questions of the vastest import in every department of neurology are awaiting solution, and the stimulus which one receives from meetings such as this furnish the impulse and renews the strength whereby we are enabled to lift fold by fold the veil from before the face of Isis, in whose lineaments we may then discover an expression of those revelations of the Book of Wisdom which we are able barely to guess at today.

Before declaring this meeting open for the transaction of scientific business I wish to thank you for the honor that you have conferred upon me in placing me in the Chair, and to bespeak your aid and coöperation in making this meeting as successful as it bids fair to be.

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CONTRIBUTION TO THE STUDY OF THE MYOSPASMS: MYOKYMIA, MYOCLONUS MULTIPLEX, MYOTONIA ACQUISITA, INTENTION SPASM.*

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There is much confusion in the classification of the various anomalies of the neuro-muscular mechanism which give rise to involuntary spasmodic movements. In a recent text-book the term myoclonia, for example, is made to include Friedreich's myoclonus multiplex, Bergeron's electric chorea, and the fibrillary chorea of Morvan, the description of the symptoms of myoclonia attempting to cover all three conditions. Again, a recent contribution describes, under the title hysterical myoclonia, a single case said to present tremor, astasia-abasia, simple chorea, fibrillary chorea and electric chorea. In other text-books myoclonia is made synonymous with myoclonus-multiplex, while still other authors do not use the word at all.

In calling attention to the persistent muscular quivering to which Schultze gave the name myokamia (from the Greek word meaning a wave), I do not propose to enter upon the general question of classification further than to clear the ground somewhat by assuming that we need not burden our minds with the effort to draw analogies between this disorder and the electric chorea of Dubini, to convulsive tic, or even to the fibrillary chorea of Morvan. Dubini's disease is infectious, endemic, and generally fatal; convulsive tic is generally unilateral, generally facial, and has little in common with the persistent fibrillary twitching under consideration, and the fibrillary chorea of Morvan is an acute disease of childhood accompanied by constitutional symptoms, in one case even by delirium, and in one case followed by death. It may appear somewhat radical, perhaps, to insist upon the absolute separation of myokymia from the myoclonus of Friedreich, to which disease its resemblance was thought by Kny to justify the name myoclonus fibrillaris

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multiplex, or from the electric chorea of Bergeron. But there is little to suggest clonic spasm in this general quivering of muscular fibers, certainly little to suggest the variety of contraction characteristic of Friedreich's myoclonus, although it is unfortunately true that the description of this disease includes occasional fibrillary twitching. The characteristic feature of the *myoclonus multiplex* consists in more or less symmetrical clonic spasm affecting the whole or greater part of the muscles involved, the muscles of the trunk and the large muscles of the extremities being most affected. Gowers[2], in discussing the various types included by different authors under the head of myoclonus multiplex, has expressed a justifiable doubt "whether any should be placed together that do not present the common features of the sudden shock-like character of the muscular contractions, their bilateral symmetry, and the comparative freedom of the extremities."

Unless, therefore, further analogy is established between myokymia and myoclonus multiplex, it seems reasonable to regard myokymia as a separate disorder.

The persistent muscular quivering to which I would call attention (best named, perhaps, myokymia), a phenomenon most frequently observed in the adult male, though a troublesome disorder, is of no grave import, as far as can be judged from recorded cases. It may run a comparatively short course and terminate in recovery, or it may persist for years without appreciable effect upon the nutrition or the usefulness of the affected muscles. It does not involve the entire muscle at any given time, but affects group after group of fibers, most commonly in the muscles of the calf and thigh, especially the glutei, less often in the shoulder muscles, and still less often in the small muscles of the hand, and those of the trunk and face.

The electrical irritability is sometimes increased to both currents; tetanus may be produced by a moderate current, and persist after removal of the electrode.

The symptom may follow lead poisoning (Buber, Karcher), or poliomyelitis (Williamson), either directly, or after an interval, and in this event its spread is apt to be among the muscles not involved in the original paralysis. It is prone to appear

during attacks of sciatica, and to persist long after the pain has passed away (Gowers, Hoffman, Bernhardt). Kny attributed it in one case to exposure and overwork, in the other to a blow in the groin. Its temporary appearance is not uncommon in health, and the fact that it may persist for a number of years without other sign of central or peripheral disease, past or present, is shown by the second of the personal cases I have to communicate.

The practical bearing of this study lies in the prognosis, which is apparently in marked contrast to that of the fibrillary twitching of progressive muscular atrophy, which it closely resembles.

Case I.—A professional man of thirty had an attack of poliomyelitis in infancy, which left the peroneal muscles and tibialis anticus permanently affected on the left side; there is a difference in measurement of about one inch in the thighs, but no local atrophy and no paralysis in this region. About six years ago he commenced to notice fibrillary twitching in the unaffected muscles of the paralyzed leg, especially in the calf muscles, which were first affected. The twitching gradually extended to the front of the thigh, and in the course of three years to the back of the thigh, particularly to the glutei. In the course of about two years the interosseus of the left index finger was attacked by fibrillary quivering. About three years ago the right calf muscles became affected, after which time quivering appeared occasionally in the muscles of the right thigh. During the past two years the intercostal and shoulder muscles have been included. There is no involvement of the face or neck.

The quivering affects for the most part only small bundles of fibers, spreading itself irregularly, sometimes passing from bundle to bundle, again, various bundles quivering at once. He is perfectly aware of the twitching through his sensation, but there is no pain. Occasionally the twitching will involve enough of the muscle to produce a movement, as of the finger or foot. The phenomenon appears while the limbs are in a state of relaxation, and is not increased by movement. It is practically constant.

There is no wasting, and the relative measurements of the legs remain the same. There is no weakness, no loss of voluntary motion, and no interference with the fine movements of the fingers as writing or handling an instrument; he is perhaps rather easily tired but no more so than can be explained by his work. There is no increased sweating, no palpitation, no dis-

turbance of sleep, digestion, or other function, the heart and lungs are normal, also the urine. He is a man of high intellectual endowment, and of somewhat neuropathic tendency.

The knee-jerk on the right side is normal, and extremely difficult to elicit on the left when the patient is tired. This diminution of reflex of the left side is of long standing and probably represents a remnant of the poliomyelitis. There is no ankle-clonus. The posterior tibial artery and the dorsalis pedis pulsate normally on both sides. He is not subject to painful cramps. This is mentioned in view of the fibrillary twitching sometimes found in intermittent claudication.

This case seems to belong to the same class with that of Williamson (which also followed poliomyelitis after a considerable interval), in that the twitching is painless, and in that it follows a definite affection of the anterior horn cells. Certain of the cases occuring after lead poisoning should perhaps be included in the same category; in the case of Buber, for example, the fibrillary twitching was painless (except occasionally at the height of an unusually extensive spasm), and it was neither identical in time nor in distribution with the paralysis; the twitching lingered long after the paralysis had practically disappeared, and was most marked in the muscles unaffected by the original process. The case of Hoffman (following sciatica), should perhaps be included in the same class, in that the fibrillary twitching persisted during the intervals of the attacks of pain. The picture in Hoffman's case is somewhat complicated by the attacks of bilateral pain in the sciatic distribution, and the doubt as to the seat and nature of the pathological process causing them, but it is significant that the quivering persisted twenty years without definite muscular alteration, the patient dying of cancer of the stomach.

The case of Karcher, in which quivering accompanied lead poisoning, is similar in every respect to that of Buber, excepting with regard to the acuteness of the attack, and it may fairly, perhaps, be classed as a milder and less lasting form of the same affection.

The case of Biancone (classed by him as mild polyneuritis) seems very similar to that of Karcher, except that the nature of the poison or infection was not apparent. The case of Mayer may possibly be included in the same group, but in the pres-

ence of spastic paralysis and in the absence of history of either recovery or of long-standing atrophy or other disturbance, its classification is somewhat questionable.

In these cases there is nothing to suggest a reflex phenomenon, and the history points so definitely to the lower motor neurone as to throw the burden of the proof upon any other seat for the disturbance. The questions (a) what portion of the motor mechanism is disturbed, and (b) what is the nature of the disturbance, are not so easy to answer and will, perhaps, never be satisfactorily answered. The prevailing view regarding the unity of the neurone as a sufferer from toxic or infectious process relieves us of the duty of deciding whether the anterior nerve cell, or the nerve fiber, is the sufferer from such process, for both are doubtless affected, if either. It is less easy to decide whether the muscular mechanism is itself affected. The discussion of this point would involve considerations too speculative to detain us.

With regard to the *nature* of the disturbance we are again in doubt as to whether the process represents an increased irritability or a decreased innervation. We have not sufficient data to justify us in discussing this question at present, and we can do little more than classify and study these cases with a view to establishing at least a prognosis for our guidance. A single suggestion bearing on this question may not be out of place.

It seems to have been assumed by those discussing these cases that we have necessarily to do with an *irritative* phenomenon, whether of direct or of reflex stimulation, but is it not equally plausible that we have to do with something in the nature of a more or less rythmical lapse from tonicity; or again, that the quivering represents something analogous to increased mechanical irritability, or to certain stages of the degenerative reaction to galvanism, namely a phenomenon resulting from a quality inherent in muscle fiber and released to a greater or less degree when the muscle is freed from the controlling influence of the cerebro-spinal axis? Viewed in this light, the comparative innocuousness and even the disappearance of the quivering would not be surprising, for neither increased mechanical irritability, nor the first stages of the degenerative elec-

trical reaction, are necessarily of grave import, though they *may* be followed by loss of function, just as fibrillary twitching may be, and generally is, followed by muscular atrophy and paralysis. It might be objected that the fibrillary quivering has no obvious stimulus for its production, and that it takes place while the muscles are at rest. We have no right, however, to assume from this that the quivering is spontaneous, for a mere change of position or of tension, a slight stretching or shortening of the muscle may well suffice to start the quivering in such anomalous musuclar tissues as those under consideration.

A discussion of this subject with my friend, Mr. John Stone Stone, the electrical expert, with a view to finding an analogy in that branch of science, gave rise to the suggestion on his part that we may have to do with a defective contact between the nerve ending and the muscle fiber, a condition known in electrical parlance as a "loose joint." This suggestion, like the theory of Duval, regarding the contact between the end brush and the nerve cell, will receive scant attention from those who find no fascination, and no reason, in the attempt to establish an analogy, if not an identity, between the electrical and the neural current, a search to be by no means hastily abandoned, however, either on account of general scepticism or even on account of apparently antagonistic findings.

The following case seems, at first thought at least, to fall into a somewhat different class from those previously considered.

Case II.—R. K., forty-eight years of age, single, a papermaker, has noticed for three years a tendency to quivering of various muscles. The onset was gradual, but the quivering soon became almost constant, and spread from the muscles of the legs and thighs to those of the forehead, the shoulder region and the lower face, all of which groups now show the characteristic fibrillary twitching with varying constancy, the gastrocnemii with absolute constancy. No pain accompanies the symptom, excepting that the patient is subject to frequent extremely painful cramps. The quivering tends to increase during movements, and shows marked increase after physical exertion. He tends to become, after walking, much more weary than he formerly did. He notices no other change in his general condition. There has been no wasting of muscles, no loss of power in any muscle or group of muscles, and no tenderness.

He has gained weight. He denies specific history; his family history as well as his previous personal history is uneventful. His parents died in advanced life, and neither had nervous or mental disease, or special neuropathic tendency. He is perhaps rather easily startled of late. He drinks two cups of coffee in the morning and drinks tea twice a day. He is subject to extremely painful cramps, generally in the thigh, occurring both by day and by night, and started by movement of the leg. The attacks last a few moments only, but may recur many times in the night. These attacks are of interest in connection with the fact that fibrillary twitching has been noted among the symptoms of claudication, or, as Dr. Paul and myself have ventured to name it, angina cruris.

Physical Examination.—There is continuous play of fibrillary twitching over the muscles of the calves, especially marked on the right; there is also quivering of bundles of fibers in the muscles of the forearm and of the shoulder group, in the chin and lip, in fact, in nearly every part of the body. The contraction has never been sufficiently extensive during my observation of the case to cause a definite movement as of a finger or toe, though he had one severe and painful cramp in the left thigh in my presence suggestive of the spasm of angina cruris. The patient is able to make all single movements, and combined movements, perfectly. The muscles of the extremities were carefully tested separately and all showed good strength. The grasps are strong, the patient can stand on his heels and toes with perfect facility, can stand with the feet together, and the eyes closed, without swaying, can ascend and descend stairs, or mount a chair with either foot with normal agility, strength and accuracy. There is no wasting of individual muscle or group of muscles; the right calf measures 14, the left 13¾ in. All forms of sensation are normal. The pupils are alike, well-rounded and of normal reaction to light and acommodation. The knee-jerks are normal and alike. The plantar reflex consists of slight flexion of all toes (normal); there is no Babinski and no clonus. The mechanical irritability in the calf muscles is increased, but tapping the muscle does not produce lasting tonic contraction.

Electrical reaction.—The application of the moderate faradic current to the muscles of the calf produces a slow tetanic contraction of the underlying bundle, persisting after removal of the electrode.* The vastus internus on the contrary, a muscle rarely the seat of twitching, reacts promptly to

*This phenomenon suggests an analogy to myotonia acquisita, but the resemblance is not sufficient to justify its classification under this head.

the faradic current with the brief and active contraction of the healthy muscle. The galvanic current presents less marked abnormality. The gastrocnemius, like the vastus internus, does not react to the direct application of this current until a strength of 10 m.a. is reached. This strength of current produces a reaction to the kathode closure slower, and more wavelike in the gastrocnemius. It was also noted that the anode closure, which caused in the vastus internus marked contraction, though less than that produced by the kathode closure, found no response in the gastrocnemius. The pulsation in all the arteries of the lower extremity is normal except in the dorsalis pedis on the right side, where it is only faintly perceptible.

In this case nothing points to affection of the anterior horn cells, either past or present. In the two cases of Kny this was also true, and he regarded the entire disturbance as functional, allied to the "Schreckneurose" of Friedreich. The rapid cure in his case lent weight to this supposition. But in our case the quivering has remained constant and unchanged for three years; other signs of neurasthenia are wanting, and it seems doubtful if we are justified in lightly turning the case off as functional, except indeed in the sense that it represents a disturbance of function of unknown pathology. It certainly seems hardly fair to class these cases under the neuroses. In the first place, fibrillary twitching, while more common in persons of neuropathic tendency, does not represent a disordered cerebral function, it is of comparatively rare occurrence in the hordes of hysterical and neurasthenic patients coming to the neurological clinics, but is of common occurrence in the well-known type of chronic anterior horn cell disease known as progressive muscular atrophy.

Is it not possible, then, that our case II and the two cases of Kny, represent a disordered function of the lower neurone allied to that established by the remains of poliomyelitis, or more acutely by the effects of lead upon this neurone but produced perhaps by faulty arterial equipment. The fact that during the progress of certain of these cases increased mechanical irritability and slight increase of direct muscular irritability to both forms of electrical current has appeared, tends to favor this hypothesis, and I do not see how we can avoid the

suspicion, at least, that we have in the quivering of myokymia a symptom analogous to, though of less grave import than the fibrillary twitching of progressive muscular atrophy. When the pathogeny of the latter phenomenon is established, we probably shall not have far to look for that of the former.

To accentuate the fundamental difference in character and distribution of the spasms seen in paramyoclonus multiplex, I append the essential points of a fairly typical case of this disease recently seen in consultation with Dr. Dow, of Lawrence.

Case III.—A married lady, twenty-four years of age, has been subject to widespread clonic spasm for eight years. The movements began in the thighs and gradually involved the trunk muscles, particularly those of the abdominal and scapular regions. Later the occipito-frontalis became involved. The muscles of the neck have shared at times, causing backward movements of the head. The family history is negative as regards this and other form of nervous disease. As a girl she was rather delicate, but not hysterical. There has been no wasting of muscles, her general strength is good, and the movements interpose no physical bar to her usual duties and pleasures. Her general health, digestion, sleep and other functions are unimpaired.

Examination shows bilateral, almost symmetrical, clonic movements. The contractions of the quadriceps femoris, for example, are practically continuous, recurring with a frequency of about eighty per minute, varying somewhat in rhythm and in force, but always affecting the whole muscle. The movements can be plainly felt in the long tendon of the quadriceps femoris by the hand even outside the clothing. The abdomen presents almost constant alternating contraction and relaxation of muscles. The oblique muscles seem chiefly involved, and the movements produced are of a peculiar twisting character, not bending the body but moving the abdominal walls. The scapulae are drawn inwards and rotated upon themselves, the lower angles approaching each other while the upper angles are elevated. The result is a sort of shrugging movement of the shoulders. The occipito-frontalis is alternately contracted and relaxed, causing movement of the scalp. The movements of the occipito-frontalis, of the scapulae, and of the abdominal muscles are less rapid than those of the quadriceps femoris. *The muscles of the hands and arms as well as those of the legs below the knee* show *no sign of spasm. No trace of fibrillary twitching is visible during prolonged observation.* The mechanical irritability is not increased.

Examination is otherwise negative. The patient is rather delicately built, but is of good muscular development and exhibits no appearance of general nervousness. She is well poised, well controlled. The knee-jerk is normal, neither lively nor disagreeable. There is no anesthesia, no sensitive point, and no other hysterical stigma.

This variety of spasm offers no suggestion of muscular freedom from central control, but suggests rather imperfect action of the central mechanism itself. It would certainly seem that attempts to class the quivering of myokymia with movements of this character and distribution will hinder, rather than advance, our study of these neurological anomalies.

The contrast between myokymia and paramyoclonus is shown, again, in a case of the latter disease recently seen in consultation with Dr. Minot.

Case IV.—A young lady of rather slender build, with tubercular process of the apieces, for several months has been subject to twitching of certain muscles, increased by excitement. Inspection shows the more or less rhythmical clonic movements to be bilateral and symmetrical, and to affect only muscles attached to the trunk. The movements are most marked in the sterno-cleido-mastoids, both of which contract together producing a slight forward movement of the head without rotation or lateral inclination. The movements are painless. Each spasmodic contraction is followed by complete relaxation; no tendency to tonic spasm appears, and no tendency to prolongation of the malposition. The patient reports a previous attack of similar nature lasting several months, in which attack the abdominal muscles were affected, and apparently the diaphragm, judging from her description of a rhythmical noise resembling hiccough. No stigmata of hysteria were found and no history of hysterical tendency elicited.

Such a case may be readily mistaken for a bilateral form of the ordinary spasmodic torticollis, and in estimating the chances of recovery without operation from that affection, cases of this nature should be excluded, for whatever the pathogeny of paramyoclonus it certainly differs fundamentally from spasmodic torticollis in clinical history, course and prognosis. I myself have reported under torticollis a case of so-called retrocollis with comparatively rapid recovery under massage and

general measures, and I am free to acknowledge that paramyoclonus was not even considered in the diagnosis.

II. Myotonia Acquisita; Intention Spasm.

With the peculiar congenial anomaly known as Thomsen's disease we are familiar, though it may not have fallen to our lot to personally observe many cases.

The characteristic picture of this disorder consists in a congenital tendency for the muscles to fall into a state of tonic contraction upon voluntary effort. The tendency gradually wears away when the effort is continued, so that the patient walks, for example, as well as a healthy individual after he has awkwardly dragged himself a short distance. The characteristic electrical reaction consists in persistent tetanus on faradic or galvanic stimulation, in the equality of kathode with anode closure, in irregular waves of contraction on application of the faradic, and rhythmical waves of contraction on application of the galvanic current. Mechanical irritability is increased, and the stroke of the hammer produces tetanus. A few cases have been reported in which a disorder similar as regards voluntary movement, and identical as regards electrical reaction has first appeared in adult life without hereditary predisposition, and an intermediate condition is recorded (Martius and Hansemann) in which the disturbance was congenital, but of intermittent appearance.

Here, again, obviously distinct cases have been classed together. Talma, for example, who first used the term myotonia acquisita, in an article proving that the myotonic reaction was not pathognomonic of Thomsen's disease, grouped together five cases, in only two of which the peculiar tonic tendency occurred independently of infectious nervous or of intestinal disorder.

In the second of Talma's cases, the muscular disorder followed influenza, was accompanied by pain, by tenderness of spine and of nerve trunks, and was temporary. In his fourth and fifth cases the tonic spasm, though increased by movement, was continuous, was ushered in by acute and violent intestinal disturbance with hepatitis, and disappeared upon the subsidence of these conditions. These two cases, therefore, bear more resemblance to tetany than to either myotonia or intention spasm.

The case of Fürstner, ushered in and accompanied by mental depression, persistent whistling respiration, vaso-motor disorders, *continued rigidity* independent of voluntary movement, showed no mechanical or electrical change, and was benefited by suggestive therapeutics. Fürstner had obviously, then, to do with a psychopathic, not a myopathic disturbance, and if the name myotonia acquisita were allowed to cover both his case and all of Talma's five cases, its application would be indeed a broad one.

Jacoby in a critical review of this subject calls attention to two essentials of myotonia, whether congenital or acquired, namely, the myotonic motor disorder, and the myotonic reaction. He reports two cases which conform to these requirements. For the simple tendency of tonic spasm accompanying or following movement, he chooses the term *intention spasm* (Seeligmüller), and reports one such case. This classification seems reasonable, but is perhaps a little too sweeping, inasmuch as it would exclude from the myotonias such cases as that of Martius and Hansemann, in which the myotonic electrical reactions were present, but the motor disorder was not that characteristic of Thomsen's disease, for the spasm did not lessen on continued effort. The following case is offered as a remarkably persistent example of *intention spasm,* accompanying diabetes mellitus.

Case V.—F. A., a patient of Dr. Phippen, of Salem, was sent by him to the Massachusetts General Hospital for consultation, and was seen in the Neurological Department, Aug. 22, 1901. He is forty-nine years of age, single, American, a confectioner. Five years ago he noticed that on walking the muscles of the leg and thigh became rigid. No fright, exposure, overwork or other exciting cause was recognized. No cramp was noticed except while walking. There was no intestinal disorder. At times he would have a feeling that if he brought his foot down squarely he would be thrown in the air, and he avoided making this movement. There has been no pain connected with the spasm though he has had occasionally a dull pain in the lumbar region. Cold does not precipitate the difficulty. The tendency to stiffness on walking gradually increased during a year, since which time it has remained stationary.

The family history, as well as the previous personal history, is negative. His father died at 67 of some throat trouble, his

mother at 37 from acute pulmonary disease. During the summer he lost twenty pounds in weight. There has been no disturbance of general health, no digestive disorder, headache, pain, tenderness or disturbance of special senses. He is not subject to cramps. There has been no shortness of breath, no swelling of feet, nor disorder of bladder or rectum. There has been no mental or physical irritability or morbid tendency, aside from the affection of gait. On one occasion he was able to run a short distance without trouble. Otherwise the condition has been unchanged.

Physical Examination.—The patient walks stiffly with a somewhat waddling gait, moderately spastic, not ataxic. The muscles of the thigh and calf, normal and soft while he occupies a sitting posture, are thrown at once into a condition of tonic contraction on the attempt to rise, and this contraction persists so long as he continues standing or walking. The tendency does not lessen on his persisting in the endeavor to walk, but remains constant throughout. He ascends better than descends stairs. He walks backward rather less stiffly than forward.

The knee-jerk is normal, rather active, alike on the two sides and not wearying on continued tapping. There is no loss of any form of sensation. The muscle and temperature senses were tested with special care, and found intact. The plantar reflex is normal (flexion); there is no Babinski. There is no clonus. There is no atrophy, no loss of motion in any muscle or group of muscles, other than the impediment interposed by the contraction. He can stand on toes and heels, and in sitting can raise toes naturally with heels on the ground. He can stand on either leg without swaying. There is no incoördination of movement in arms or legs. He has great difficulty in stooping. The muscles are of good size, but nothing in their appearance suggests hypertrophy. The left thigh measures 14½ inches, the right 14⅜ inches, in the smallest part. The muscles of the thigh are well rounded and of normal appearance, those of the calf are well developed, but not distinctly hypertrophied; the calves measure each 14¼ inches. There is no increased mechanical irritability of any muscles of the leg, and no change in electrical reaction, unless, perhaps, the reaction to the faradic current is rather slow in the gastrocnemius, not sufficiently so, however, to suggest tonus.

The urine, which was found normal at the last examination (1 year ago), at this examination contains sugar. The specific gravity is 1024, the reaction acid, there is no albumin. Further examination of the urine has shown that the presence of sugar is constant, though without constitutional signs of diabetes.

The symptoms in this case differ from those of myotonia* in that the tendency to tonic contraction does not wear away upon continued use of the parts, and in the absence of the myotonic electrical reaction. Inflammatory and degenerative disease of the nervous system, peripheral or central, is eliminated by the absence, during five years, of constitutional symptoms, the absence of disordered reflexes, nutrition, sensation, vaso-motor or secretory mechanism, as well as lack of tenderness, of pain, of pupillary change, and of motor disorder, other than the impediment offered by the tonic contraction. We have obviously to do with a non-inflammatory, non-degenerative and non-infectious muscular affection without known organic basis. Nor are we justified in classing the case as hysteria or neurasthenia, excepting as we may choose these designations to cover all nervous disorders without recognizable organic basis, in which case we are no nearer to understanding the phenomenon than if we had not named it at all. No attempt has been made to solve these cases, and no basis has been suggested, beyond that of increased irritability, which really means nothing. Here again the study offers at present little satisfaction beyond the establishment of the prognosis, which is apparently good, as regards life and general health, unless other disorder is present. It is too soon to establish a prognosis as regards the disappearance of the symptoms. Jacoby's case apparently recovered in less than a year. It is to be hoped that all similar cases will be reported, that we may have reliable data upon the question of the course and ultimate result of the malady.

To revert to the analogy between the neural and the electric force, it may not be without the realm of plausibility to suggest that if such analogy is ever established it may appear that the symptom under consideration is due to something akin to the residuary magnetism of which we have a practical

*Myotonia appears to have been used also in the collective sense. Hochsinger describes as myotonia of infancy the persistent hypertonicity of the flexors, and diagnosticates it from tetany, but if myotonia is used as an inclusive term it is a question if it should not cover tetany itself, while if it is used in the narrow sense it has been preëmpted by Thomsen's disease. Such considerations render it advisable to avoid using myotonia as well as myoclonia as generic terms, substituting, perhaps, the word *myospasm*, clonic and tonic (a suggestion for which I am indebted to Dr. Courtney).

demonstration in the occasional tendency for the vibrator of the faradic apparatus to remain glued to the electro-magnet.

In conclusion, it seems pertinent to make the following suggestions:

(1) The term *myoclonia,* as a collective designation for the unrelated disorders, should be discontinued.

(2) The term *myokymia* should be limited to cases showing, without hereditary or congenital history, widespread muscular quivering, without atrophy or other indication of progressive degeneration of the nervous system, without constitutional symptoms, and without sign of present infectious or other acute disease. Cases should not be excluded, however, on account of preceding or introductory symptoms pointing to disease of the lower neurones, if such disease has either disappeared or come to a standstill.

(3) The term *myoclonus fibrillaris multiplex* should not be applied to such cases, since it suggests a relationship between myokymia and the paramyoclonus multiplex of Friedreich, with which it has nothing in common.

(4) *Myoclonus multiplex* (the prefix para seems superfluous), should be used to designate bilateral clonic spasms involving whole muscles or groups of muscles, generally those attached partly or entirely to the trunk.

(5) The term *myotonia acquisita* should be limited to non-hereditary and non-congenital cases in which otherwise healthy individuals present the typical motor disorder or the typical reactions of Thomsen's disease. This term should not include the rigidity accompanying marked intestinal disorder or pronounced psychopathic states, even though the rigidity in the latter conditions may be increased by voluntary movement.

(6) The tendency to spasm on attempted voluntary movement, unless accompanied by the typical motor disorder, or the typical reactions of Thomsen's disease, should be classed as *intention spasm.*

(7) The term *myospasm,* clonic or tonic, may be used instead of myoclonia and myotonia, when it is desirable to include under one head the various forms of involuntary muscular contraction without known organic basis.

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UNUSUAL CHOREIFORM ALTERATIONS IN THE WIDTH OF THE PALPEBRAL FISSURE OF BOTH EYES, OCCASIONED BY SPASM OF THE LEVATOR PALPEBRAE MUSCLES.*

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In October last, O. W., a colored boy, eight years of age, was brought to my service at the Howard Hospital, on account of a curious tendency to raise his upper lids. His mother attributed these movements to an accident which he had received during the summer months, in which the back of his head had been run over by a wagon, for she said that prior to this the boy had never been troubled with his eyes nor had she noticed anything unusual about them. At the time of examination he complained somewhat of headache but especially of a feeling of sand in his eyes.

The movements of the eyelids were very curious and striking, owing to the alterations in the width of the palpebral fissure which they occasioned and the amount of sclera which became exposed, the negro skin contrasting vividly with the pearly white of the sclera. The contractions were clonic and rythmical, the fissures widening about fifteen to twenty times in the minute; they were entirely under the control of the will, the patient being able to initiate them or to cause them to cease when commanded to do so. The excursions of both eyes were normal; there was no nystagmus; vision and accommodation were normal; both eyes were moderately hypermetropic.

It was easily apparent that the widening of the fissure was occasioned by a contraction of the levators of the lids, and that this was accomplished without the assistance of the frontalis, the eyebrows remaining perfectly quiescent: it was further patent that the movements had no connection with the act of winking, as the contraction of the orbicularis occurred in the

*Read before the Ophthalmological Section of the College of Physicians of Philadelphia, April 15, 1902.

usual way, entirely independent of the widening of the fissure. The question then arose, which part of the levator was acting, the bulk of the muscle, that controlled by the third nerve, or the smaller portion, consisting of unstriped fibers, which is innervated by the sympathetic. It seemed probable that it was the contraction of the former that occasioned the widening, for it was thought that if it were due to a spasm of the muscle of Müller, changes in the size of the pupil, the iris being likewise under the control of the sympathetic, would probably have been observed also, whereas the pupils, which were 3 mm. in size, remained unchanged during the dilatation movements. It is of interest to note in this connection that the Graefe lid sign was absent and that there was no exophthalmos.

The pathogenesis of the affection was assumed to be choreiform, the contraction of the levator replacing the contraction of the orbicularis, which is so common a manifestation of habit chorea. As is so often the case in chorea of the lids, there were no associated twitchings in the muscles of the face, neck or elsewhere. Spasm of the levator is a very rare affection, and notwithstanding the many and bizarre forms of tremor and spasm of ocular and associated muscles of the face and neck which have been recorded in chorea, its occurrence in connection with this disease has not been remarked before. An instance of primary tonic cramp in an insane patient has been recorded by Pick, in which the Graefe lid sign was present, although there were no signs of exophthalmic goiter: in most of the recorded cases of spasm of this muscle, however, there have been other associated ocular conditions. Thus Wilbrand and Saenger in their recent and comprehensive work upon the neurology of the eye, mention in this connection cases of Salzmann and Rampoldi, where there were rythmical changes in the width of the palpebral fissure synchronous with pupillary contraction. The same authors report cases seen by Hutchinson and Gooding where the spasm of the levator was induced reflexly by diseased teeth. Finally, they give the notes of a series of cases where the spasm was probably due to irritation of the nuclear region of the oculo-motor, being associated with paresis of groups of muscles supplied by this center.

Although under ordinary conditions the action of the levator in raising the lid is inseparable from a contraction of the frontalis, it is possible by practise to innervate the levator alone, and to widen the fissure without bringing the frontalis into play. I have seen this isolated action of the levator, though but to a slight degree, in several patients with chronic conjunctival irritation, where the movements of the lid seemed to bring momentary relief to the local sensations, and I have repeatedly noticed the fissures widen independent of any action of the frontalis when patients fixed the finger in the cover test at the near point; the widening in the latter instance being attributable to an associated stimulation of both internal rectus and levator muscles.

The course and conduct of the case were simple, the movements in the lids disappearing as soon as atropine was instilled for refraction purposes, and no return of them has been noticed since the proper glasses were worn.

INVOLUNTARY MOVEMENTS IN A CASE OF ATAXIA.*

By John H. W. Rhein, M.D.,

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The purpose of this brief note is not so much to record an unusual symptom in tabes, for involuntary movements in this disease are more frequent than one is led to believe; but to call attention to the failure on the part of English and American observers, with few exceptions, to mention these phenomena in their descriptions of the symptoms of ataxia.

The patient, a widower, a gambler by occupation was 52 years of age. His life by reason of his vocation, was an irregular one as to hours and eating, but he had always been temperate in alcohol which is usual with his class, and never suffered from syphilis. In the use of tobacco he was always moderate. He had never had any serious sickness.

His family history was negative as far as it could be elicited. His father died of typhoid fever, his mother of senility at 83 years of age, one brother died of some stomach disorder, one sister of an unknown disease, and he had one brother living and well. He had no children.

His disease began three and one half years prior to his first visit to my office. The first indication of his infirmity consisted of a sense of restlessness; sharp pains in his legs, and a tightness around the chest as though a rope were bound tightly about him. His walking became insecure in six months' time, and gradually grew worse. Early in his disease he lost his sexual power, and complained of double vision.

In the few months prior to his first examination, he had had difficulty in emptying his bladder and frequent catheterization was necessary. He also developed about the same time severe pain in the rectum, which came in attacks lasting intermittently for some hours to two or three days at a time. His bowels had been constipated from the beginning of his disease.

For two or three months he had suffered from a cough associated with a greenish yellow sputum, and this was growing steadily worse.

Upon examining him in November of 1899, I found him

*Read before the Philadelphia Neurological Society, April 22, 1902.

generally emaciated and weak. He could walk with the assistance of a cane, but was very ataxic. He could stand but a few seconds with his feet close together, and if he closed his eyes he fell at once to the ground. His knee-jerks could not be elicited in the usual manner nor by reinforcement. His plantar reflex was present. The arm-jerks could not be developed.

He confused hot and cold sensations in places in both arms and legs. Pain sense was diminished but sensation to touch was good everywhere, while both were delayed in arms and legs and in an irregular distribution on the trunk.

There was no evidence of spinal atrophy. The tongue was smaller on the left side than on the right, but I was unable to conclude that there was any real hemiatrophy.

He complained bitterly of the sharp lancinating pains which were chiefly situated in the legs, but were at times felt in the chest wall and more rarely in the arms. His heart was normal. Examination of his lungs disclosed beginning consolidation.

Dr. de Schweinitz examined the eyes and reported as follows: Semi-dilated pupils, the left being the larger; typical reflex iridoplegia with moderate contraction on the effort of accommodation. In each optic disc there was a low-grade congestion with beginning degeneration in the deeper layers. The veins were unusually large, the arteries normal in size. The right external rectus muscle was paralyzed 25 degrees. With suitable correcting lenses vision was normal. The form-field was slightly contracted peripherally; the red field, especially on the left side, distinctly contracted. The other color-fields were not taken.

I have reserved till the last to describe the symptom which was of special interest. There was observed in the legs, especially in the right, a clonic spasm of the extensors of the toes. All of the anterior tibial group of muscles seemed to be involved. I could not observe any spasm of the posterior muscles whatever. These contractions were most distressing though painless. They were very different from the sudden violent contraction of the leg muscles common in ataxics which are often associated with sharp pain.

The spasm lasted irregularly for hours and appeared more frequently during the night. There were days when there was no spasm whatever. There was a possible relation to the onset of bad weather, though I was never absolutely sure that this was true. The contractions were slow, about two a second, rythmical and mild. Four to ten contractions were observed,

then a brief interval of rest, then a few more contractions and so on for an hour or so, after which as I have stated a long interval, often days, would elapse when they were entirely absent. These movements began about three months before death.

I was surprised in reviewing the literature of involuntary movements in ataxics to find so few references to this subject in the recent publications, especially in the English language.

Many of the systematic writers have failed to even mention this symptom in the descriptions of ataxia, while only brief mention of this condition is found in Church and Peterson, Hirt, Oppenheim, Raymond and Marie.

This prenomenon was described as early as 1840 by Cruveilheir[21] and later by Leyden[1] (1863); Trousseau[2] (1865); Rosenbach[3] (1876); Grasset[4] (1878); Berger[5] (1880); Audry[6] (1887), and more recently by Laquer[13] (1890); Rossalimo[14] (1893); Raymond[16] (1894); Marie[17] (1895); Hirschberg[20] (1897); Oppenheim[23] (1898), and others.

Trousseau[2] described this condition as being a sudden twitching of the muscles. Later observers described the movements as being athetoid (Raymond, Laquer, Berger), and as choreiform and athetoid (Rossalimo and Audry). Rossalimo uses the word "amyotaxia" to describe this condition.

Audry's conclusions were as follows: First, In exceptional cases there is present in ataxics, choreiform and athetoid movements. Second, These movements are not an exaggeration of the spontaneous twitchings in the extremities, but a condition differing absolutely from ataxic movements. Third, They are accompanied by contractures and are due to irritation of the lateral columns.

Hirt[15] also believes that these movements are indicative of irritation of the motor columns. They, however, lack the spasmodic character of athetosis, and the contractions are less quick and irregular.

More recent writers, however, notably Stern[9], Marie, Oppenheim and Hirschberg are of the opinion that they are simply manifestations of incoördination.

We may divide the involuntary movements of tabes into (1) The associated movements, which may exist also in com-

bined system disease of the spinal cord, cerebral hemiplegia, spastic spinal paralysis and cases of unilateral lesion of the spinal cord (Strümpell[10], Stintzing[8]). (2) Sudden twitching of the trunk or extremities which may occur with or without pain, and are more frequent at night. (3) Twitchings in isolated muscles or parts of a muscle. (4) Fibrillary twitchings. (5) Rythmical tremor resembling that of paralysis agitans (Hirt[15]). (6) Passive movements, which Hirschberg describes as being those movements of the legs or thighs which occur when a patient who is lying down attempts to sit up in bed without the assistance of his hands; or when he coughs (Stinzing), and (7) finally involuntary movements previously described as athetoid movements.

These movements are most frequently seen in the hand and fingers, though they are present also in the toes and feet, in the muscles of mastication and in the tongue. The little and ring fingers are usually first to be involved, but the middle finger may be involved, or some movement of the hand such as rotation, supination, fiexion or extension may be the first movement to be observed.

They are involuntary, but may be partially controlled by great effort of the will, or by support.

Hirschberg has studied the subject very carefully and concluded that the condition is less rare than it is usually supposed to be, and that it can not be considered a complication of tabes, but rather a particular manifestation of motor incoördination.

When the eyes are closed these movements become more marked, in fact early in their development they may be apparent only when the eyes are closed.

Note:—Since reporting this case, I have seen a second case in which the fingers of both hands and the toes of the left foot were involved. The movements were slower than in the case cited above and were only present when the patient closed the eyes.

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PHILADELPHIA NEUROLOGICAL SOCIETY.

March 25, 1902.

The President, Dr. John K. Mitchell, in the chair.

Adiposis Dolorosa.—Dr. Max H. Bochroch presented a typical case of this disease.

Adiposis Dolorosa.—Dr. F. S. Pearce exhibited a case of adiposis dolorosa of the diffuse form in a negro woman forty years of age. The patient had begun to gain fat rapidly after the birth of her second child, twenty years ago. The woman reached a weight of 303 pounds and now weighs 295 pounds. The woman had not complained of subjective pain nor had she the typical tenderness on pressure, but the deposit of fat was typical of the disease, and there was the feeling as of "bunches of worms" on palpation. The wrists and ankles were free, but there were large masses of fat over the upper arm, the upper outer aspect of the thighs, the scapula and abdomen, which hung in heavy folds. There were no mental symptoms. She did considerable hard work and her general health was excellent. The patient had been referred to Dr. Pearce for diagnosis by Prof. Jas. M. Anders. While there was absence of subjective symptoms or disturbance of the mind, yet with the typical picture of fatty deposits the diagnosis seemed plausible. Forel has recently reported cases of adiposis dolorosa without pain or tenderness.

Dr. A. O. J. Kelly inquired of Dr. Pearce in what way his case differed from one of ordinary obesity.

Dr. D. J. McCarthy, with reference to the case shown by Dr. Pearce, said that the deposit of fat did seem different from that seen in ordinary obesity. In the upper part of the arm it seemed of a fibroid character. In discussing this subject the tendency is to run to extremes and classify all classes of pathological fat as adiposis dolorosa. In adiposis dolorosa distinct, painful lumps of fat can be felt. In this case there was no tenderness. We must differentiate between obesity and these conditions of localized neuritis in fat. There is probably a difference mainly in the chemistry of the fat rather than a difference in the condition of the fat.

Dr. William Pickett remarked that the name Dercum's disease or adiposis dolorosa is given to a symptom-complex. He thought that Dr. Dercum had so far not committed himself to the idea that this is a distinct entity. It is very likely that there are numerous cases ranging from acromegaly and myxedema to these cases of special form of obesity with neuritis. It may be that ordinary obesity belongs in this series. If, however, the name Dercum's disease is to mean anything, it must be restricted to those cases in which there is not only fat in localized deposits, but also tenderness in these masses. This does not invalidate the claim of Dr. Pearce that this case is related to the disease described by Dr. Dercum.

Dr. F. S. Pearce said that while this was not a typical case of adiposis dolorosa, yet he thought that it was allied to adiposis dolorosa. The physical character of the fat and its distribution seemed to point to this diagnosis.

Dr. J. K. Mitchell remarked that he thought it should be emphasized that almost every case of excessive fatty deposit presented varying spots of tenderness, often in fixed positions, but that did not make them cases of adiposis dolorosa. They have not the characteristic sym-

metrical fatty deposit found in Dercum's disease, where the method of deposit as well as its character has to be taken into consideration.

Primary Degeneration of the Pyramidal Tracts.—Dr. Wm. G. Spiller read a paper on this subject (See page 265).

The Muscle-factors of Ankle-clonus.—A paper on this subject was read by Dr. S. Weir Mitchell (See page 257).

Dr. William G. Spiller inquired whether or not in those cases in which clonus was obtained when the leg was extended on the thigh, the gastrocnemius took any part in the movement. When the leg is extended it is more difficult to obtain clonus, but in some cases, possibly where the extension is not complete, it can be developed.

Dr. A. A. Eshner said that if the leg is partially flexed in the ordinary way in which ankle-clonus is obtained, the gastrocnemius is relaxed to such a degree that it takes no part in the movement. This means that if the gastrocnemius takes any part when the leg is extended on the thigh this is not essential, as it does not enter into the movement when the leg is partially flexed. At Dr. Mitchell's request he had studied the relations of these muscles on the cadaver. Both of the muscles are supplied from the same nerve (internal popliteal) and the filaments for each come off close together. Whether as a result of mechanical factors or not it is a fact that the soleus is the essential agent in ankle-clonus. The soleus and the gastrocnemius together can be looked upon as a three-headed muscle with a common tendon, and as with other many-headed muscles one part may be set in action independently of others, and in accordance with their different points of origin.

New Diagrams of the Zones and Centers of the Human Cerebrum Based upon some recently-acquired Facts and a Study of Flechsig's Association Centers.—These were exhibited by Dr. Charles K. Mills.

Dr. William G. Spiller referred to the possibility of the second temporal convolution being the cerebral representation of the vestibular nerve, as Dr. Mills had suggested. There is nothing in anatomy pointing positively to that. Dr. Flexner and Dr. F. A. Packard had sent the speaker a brain in which the first and second temporal convolutions were intensely atrophied. The external bundle of the peduncle, which is believed to come from the second temporal convolution did not appear to be degenerated in this specimen. It is possible that the second temporal convolution contains fibers transmitting impulses upward, and that it is the representation of the vestibular nerve, but this remains to be proved.

Dr. D. J. McCarthy described Flechsig's method of investigation. So far as the developmental fibers are concerned he works with fetal brains of different periods of development, and serial sections of the entire brain are cut both in the horizontal and vertical directions. He has some twelve or fifteen thousand of these brain sections. For years Flechsig has insisted that in the study of mental conditions this method of serial sections is the only correct one to follow. His studies have been continued over a great length of time, and an article of two or three pages may represent the work of four or five assistants for two or three years.

Two Unusual Forms of Clonus.—This paper was read by Dr. John K. Mitchell (see page 260).

Dr. F. S. Pearce had had an opportunity to examine one of the cases referred to by Dr. Mitchell. He suggested that possibly one reason that there was easily excited lateral movement in the ankle was to be found in the condition of the joint. He thought there was sub-ankylosis at

the ankle and as a result the lateral movement appeared better than that between the tibia and astragulus.

Dr. John K. Mitchell said that the patient had marked ankle-clonus, very violent and long-continued. As far as he knew there was no special limitation to movement in the ankle joint.

Brachial and Pectoral Reflexes.—Drs. Mills and McConnell exhibited a patient showing certain unusual brachial and pectoral reflexes. Tapping over the inner aspect of the shoulder and also at the point where the second rib joins the sternum caused certain reflex movements in the fingers. They had found the same reflex phenomena in several other cases, and in these cases the other tendon and muscle phenomena so well known in the upper extremity were found.

Dr. D. J. McCarthy suggested that in tapping over the inner aspect of the shoulder the long head of the biceps was tapped, giving rise to a tendon-jerk, or the muscle-muscle-jerk of S. Weir Mitchell.

Dr. William Pickett said that in his study of the scapulo-humeral reflex, he had described a motion of the arm similar to that in von Bechterew's reflex, from tapping the point of the shoulder. It is barely possible that Dr. Mills' reflex is a modification of the latter, and that both are modifications of the von Bechterew reflex. The muscles implicated by tapping at any of these points are numerous and often far distant from the point tapped. He had a number of times observed the pectoralis major contract quite strongly on tapping at the inner edge of the scapula. There are analogies for this distant action of the muscles. Drs. Mills' and McConnell's observation he considered an extremely interesting one.

Periscope.

Deutsche Zeitschrift f. Nervenheilkunde.

(1902. Vol. 21, Heft 1-2.)

1. Multiple Sclerosis of the Central Nervous System. Hoffmann...
2. Pathological Anatomy of Hemiathetosis, at the Same Time Contribution to the Knowledge of the Tracts Descending from the Region of the Corpora Quadrigemina. Haenel.
3. Five Cases of Tumor of the Cerebellum. Von Foss. .. ..
4. Contribution to the Knowledge of the Symptom Complex of Disseminated Sclerosis of the Posterior and Lateral Columns, Based upon the Changes Found in a Case of Meningomyelitis, Probably Luetic. Bikeles.
5. Clinical and Experimental Studies upon the Innervation of the Bladder, Rectum and Genital Apparatus. Müller.
6. Book Reviews.

1. *Multiple Sclerosis.*—Hoffmann contributes a careful analysis of the literature of multiple sclerosis, partly illustrated by his own experience—which includes the observation of more than 100 cases—and three microscopical studies of the central nervous system. The etiology is not clear. The disease occurs most frequently between the ages of 18 and 35, equally in men and women, most frequently among the laboring class, and occupation appears to be without particular influence. Hoffmann is not inclined to lay great stress upon the hypothesis that the disease follows infection. In his own cases infectious diseases preceded the symptoms in 5 per cent. of the cases. Syphilis plays no part. The metallic poisons have been supposed to have influence; in his 100 cases there was but one in which the occupation brought the patient into contact with lead. Alcohol, dissipation, etc., do not appear to play any particular part. Exposure to cold, especially associated with dampness has been supposed to play an important rôle. In Hoffmann's cases there were 13 who gave such an history, but only 4 in which some other etiological factor was not accused. Emotional disturbance, parturition and injury have also been supposed to play important parts. Hoffmann is inclined to believe that they tend to make the disease worse when it actually exists, although in his 100 cases 13 gave the history of severe injury. However, in more than one-half of all cases no etiological factor can be determined. The symptomatology of the disease is exceedingly variable. Mentally there is diminution of intelligence, and perhaps symptoms simulating paralytic dementia. Occasionally there are attacks of Jacksonian epilepsy. The speech defects are scansion, dysarthria, aphasia, etc. The motor defects consist of hemi-pareses, paralyses, mono-paresis or paralysis in the distribution of a single motor nerve. The patients may have palpitation of the heart, vomiting, dyspnea, glycosuria and polyuria. The symptoms may simulate those of bulbar paralysis, or there may be symptoms of paralysis agitans, including vertigo, pro- and retro-pulsion, etc. In 54 per cent. of his cases Hoffmann observed disturbance of speech; in 64 per cent., vertigo; in 6 per cent. there were gastric crises associated with vomiting. There may be disturbance of sight involving one or both eyes, disturbance of the eye mus-

cles. There may be swelling of the papillæ observed by Hoffmann in 50 per cent. of his cases. Nystagmus was present in 56 per cent. of his cases. Ataxia of the iris or intention tremor of the iris has been described as a rare symptom. Disturbance of smell and taste are rare; disturbance of hearing is slightly more common. The spinal symptoms are particularly those involving the motor tracts. They are usually spastic, although flaccid paralysese with loss of the tendon tremor may occur. Intention tremor occurred in 71 per cent. of Hoffmann's cases. Swaying when standing with the eyes open is also fairly common. Various symptoms of paresthesia occurred in 66 per cent. of Hoffmann's cases. Objective hypesthesia with anesthesia, and occasionally disturbance of the stereognostic sense are also frequently present. In the majority of cases the tendon reflexes are exaggerated. The skin reflexes vary considerably. The abdominal reflex was absent in 29 per cent. of Hoffman's cases. Babinski's reflex is frequently present. In 60 per cent. of Hoffmann's cases there was disturbance in urination. Impotency occurred in 2 cases; conception was apparently not affected. Vasomotor changes are rare; muscular atrophies are also rare, but do occur. The course is variable. From time to time patients have remissions that usually are followed by a fresh increase. The diagnosis in the early stages is excessively difficult. Hoffmann mentions 23 other diseases that may be confused with multiple sclerosis. Cure is practically unknown, at least it is not certain. The prognosis is therefore unfavorable. The duration of the disease varies from 2 months to 20 years. Treatment consists in avoidance of other injurious factors, rest, moderate hydrotherapy, and the administration of certain drugs, particularly quinine, potassium iodide, nitrate of silver, strychnine, antipyrin, salicylic acid, ergotin, arsenic, etc., bath cures of various kinds, and mercurial inunctions, which in Hoffmann's experience usually make the case worse. The pathological anatomy is variable. There are changes in the vascular system, in the neuroglia and in the true nervous elements. No satisfactory explanation of these changes has been given. The majority of authors believe that the process is inflammatory; a few look upon it as a form of gliosis, perhaps congenital in type. Hoffmann does not believe that we are in a position to determine in which of the elements of the central nervous system the process actually arises.

2. *Hemiathetosis.*—Haenel reports the case of a man twenty-one years of age who had always been paralyzed on the left side. At the age of twenty he had contracted syphilis, and some months afterward developed cough, expectoration, emaciation and night sweats. The physical examination showed no motor nor sensory disturbances excepting slight sluggishness in mimicry on the left side. The left arm was somewhat smaller, kept in a position of slight flexion, and in continual athetoid movements. It was possible to control these only for a brief interval of time. The muscles were spastic; the tendon reflexes could not be elicited; the left leg was also atrophic. There was pesequinovaris, no athetosis, and fair control of voluntary movement. The patellar reflex was weak on the right side, and absent on the left side. Sensation was normal in both arms and legs. The patient had a tuberculous cavity in the lung from which he died. The autopsy showed the presence of an old focal lesion in the subthalamic region which had produced almost complete destruction of the right crus, and which extended from the right internal geniculate ganglion as far as the surface of the posterior corpora quadrigemina. It appeared to have been a vascular rather than an inflammatory process. The pyramidal tract for the left side of the body was absent. There were however, certain groups of fibers not normally present that could be distinguished in the dorsal portion of the

pyramidal tract in the medulla, particularly two groups which Haenel describes very carefully. He believes that there can be no doubt that these fibers were newly formed on account of their situation and general appearance. The source of these fibers was not clearly made out, but it is possible that they came from the pyramidal tract belonging to the other side. The case appears to prove the contention of Bonhoffer, that in cases of athetosis the subthalamic region must be involved. It was difficult to understand why atrophy should occur, although it is known that it almost invariably does occur, and it is also difficult to understand why spasticity was not present in the legs. No atrophic condition was found in the brain cortex.

3. *Tumor of Cerebellum.*—Von Foss reports five cases of cerebellar tumor. The first, a woman thirty-five years of age, a year before her death began to have severe headaches. On one occasion she became unconscious and was paralyzed in three extremities. Later these three paralyzed extremities frequently showed clonic convulsions. She developed vomiting, gradual loss of vision, her gait became uncertain, there were athetoid movements in the right hand and ataxia of the left hand with loss of the stereognostic sense. Argyll-Robertson pupils were present, and from time to time attacks of complete paralysis of one or more extremities. A diagnosis of cerebellar tumor was made and an operation performed, 4 days after which the patient died. At the autopsy a tumor was found in the posterior portion of the fourth ventricle with marked dilatation of the central canal, in the cervical position of the cord. The tumor was an angio-sarcoma. The second case, a woman 34 years of age, whose sight became bad about three months before admission to the hospital. There was nystagmus, some paresis in the right shoulder, diminution of sensation in the distribution of the left trigeminus, and persistent headache. Mercurial inunctions did not produce any improvement. The patient died and at the autopsy a tumor was found springing from the anterior surface of the right tentorium. Microscopically it was an endothelioma which could have been readily removed by operation. A similar tumor was found in the liver. The third case, a man twenty years of age, had had headaches for two years, which had developed two years after a severe fall from a horse. He was drowsy, appeared somewhat demented; there was enlargement of the cervical glands posteriorly, diminution in vision, inequality of the pupils, no paresis, slight ataxia of the extremities, and disturbance of gait. The patient improved somewhat on antisyphilitic treatment. The fourth patient, a man nineteen years of age, had headache for about 6 months. Towards the end of this period his vision diminished and he became finally completely blind. There was also deafness in the right ear, evidently of nervous origin, loss of smell on the right side, and the muscles of the lower extremities were hypotonic. The reflexes were lost with the exception of the Achilles tendon reflex; sensation was normal. Lumbar puncture was negative. The patient from time to time had attacks of vertigo and left the hospital, dying a short time afterwards. No autopsy was obtained. The fifth patient, a man 21 years of age, for five years an excessive drinker, had an attack in which he lost consciousness, then had severe headaches, vertigo tintitus, weakness of the legs and vomiting. His mind became somewhat weaker, there was diminution of vision, irregular gait, normal tendon reflexes and increased skin reflexes. Slight hyperalgesia and slight albuminuria were present. The patient improved in his symptoms upon potassium iodide, but the motor disturbances grew worse and he died. Autopsy was not obtained. Von Foss gives a valuable table of the symptoms of these five cases, and some of these symptoms are of considerable interest. For instance, the giving way of

the legs, the atypical disturbance of gait, and the rapidly fatal course of the two last cases. In three of these cases there was rigidity of the muscles of the neck, a symptom upon which Oppenheim lays particular stress.

4. *Disseminated Sclerosis.*—Bikeles reports the case of a man forty-five years of age, who at the age of twenty had syphilitic infection. From that time he was healthy until a few months before his death, when he had severe pains in the lower portion of the abdomen, sometimes associated with eructations and vomiting. There was difficulty in the retention of urine, some disturbance of gait with a feeling as if he were walking on cotton. There was obstinate constipation, and finally as a result of repeated catheterization, cystitis. There was some loss of power in the lower extremities, some diminution of pain sense in the same region, and the knee-jerks were distinct. Otherwise the reflexes were normal. The patient finally died of the cystitis, which had become complicated by a pyelonephritis. The essential changes found in the central nervous system were: an area of degeneration in the whole circumference of the spinal cord, areas of perivascular sclerosis in the lateral and posterior columns, with secondary ascending and descending degeneration. In the brain there was slight leptomeningitis, and in the medulla, in addition to this, some perivascular sclerosis. The essential clinical symptoms were paresis without spasticity, preservation of the knee-jerks associated with symptoms of degeneration in the posterior columns. The case resembles somewhat the symptom complex described by Rothmann, but differs in the fact that there were some pupillary disturbances, that is, the pupils were contracted and reacted sluggishly to light. The character of the pathological changes was strongly in favor of a luetic process.

5. *Genito Urinary Nerve Supply.*—Müller contributes a long paper upon the innervation of the bladder, rectum and sexual organs. He reports a remarkable case of a man forty-eight years of age suffering from transverse myelitis, in which every three or four hours the urine was spontaneously discharged without any control by the patient's will, and in the intermediate no dribbling occurred. A somewhat similar case was observed in a woman who also had some transverse lesion in the dorsal portion of the spinal cord. In a man who had an old transverse myelitis of 16 years' standing a similar peculiar condition existed, and in another, a man thirty years of age suffering from multiple sclerosis, the urine also was spontaneously evacuated. In a girl suffering from the same disease urination occurred spontaneously not more than once or twice a day, and catheterization showed the presence of a large quantity of residual urine. In a patient suffering from disseminated myelitis, who as a result of repeated catheterization developed cystitis, the urine was discharged in moderate quantities every hour. There was also spontaneous evacuation of the rectum. Diseases of the conus do not appear to interfere with the urinary functions, but it is interesting to note that in diseases of the lower portion of the spinal cord the same phenomena may ensue, and therefore Müller is convinced that the center for the bladder and rectum lies external to the spinal cord. He mentions a number of cases of locomotor ataxia in which there were marked disturbances in urination and in the sexual functions. In these cases there is evidently some anesthesia of the bladder and rectum. He mentions 2 cases of combined systemic disease in which there were disturbances of micturition; that is to say, the bladder was evacuated involuntarily at frequent intervals throughout the day. Similar symptoms may occur in cases of hysteria. He has also performed some experiments upon dogs which consisted essentially in the removal of larger or small-

er portions of the spinal cord. The results confirm Müller's opinion that the functions of the bladder and rectum are vegetative in character, and are innervated chiefly from the sympathetic nervous system. The article contains some very valuable data regarding the investigation of the disturbances of these functions in diseases.

J. Sailer (Philadelphia).

Centralblatt für Nervenheilkunde und Psychiatrie.

(1902. March 15.)

1. Experiments with Non-isolation Treatment and Hydrotherapeutic Measures. W. Alter.
2. Embarrassment under Observation. von Bechterew.
3. New Toxic and Therapeutic Properties of the Blood-serum of Epileptics and their Practical Applications. Carlo Ceni.
4. Comments on F. Nissl's Paper: "Hysterical Symptoms in Simple Mental Disturbances." Ernest Storch. Reply by F. Nissl.

1. *Non-isolation and Hydrotherapy.*—In the Leubas Asylum it has been the custom to isolate many of the chronic insane on account of violence, etc. Alter has succeeded in managing these cases in open wards through the systematic use of prolonged baths and packs. At first patients were kept in the bath at a temperature of 34° to 36° C. from 1½ to 3 or 4 hours; later in some cases the period was extended to 12 or 14 hours. Sometimes preliminary and adjuvant doses of paraldehyde, trional or chloral were employed to render the subject more tractable, but soon it was possible to dispense with drugs, the patient gaining in weight, becoming quiet and sleeping well.

2. *Embarrassment under Observation.*—Von Bechterew describes three neurasthenics in whom palpitation of the heart, confusion, weakness of the limbs, etc., lasting about five minutes, were prone to come on when anyone "caught the eye" of the patient. Bechterew formerly reported such a case in which this symptom of "Blickschen" coexisted with "forced laughter."

3. *Blood-serum of Epileptics.*—Ceni claims to have begun simultaneously with D'Abundo (1898) his study of the question whether the blood-serum from a bad case of epilepsy can modify the condition of a milder one. Of six epileptics studied, two showed symptoms of true poisoning from the injections, while in the others no effect was discerned. Ceni concluded from this that the blood-serum of epileptics contains no antitoxic substance in the true sense, but does contain a special poison. Whether the resistance of the organism to this special poison can be increased, perhaps to the point of rendering the subject immune to its effects, was then studied in two ways: first, the blood-serum of one epileptic was injected into another in increasing doses; second, a patient's blood-serum was re-injected into his own circulation some days later. Ceni was able to answer the question in the affirmative from the results of each of these methods. Of eight epileptics thus treated two showed toxic effects, but the others were greatly improved both as regards the motor manifestations of their disease, and as regards psychic and sensory symptoms, so that an actual cure may be hoped for in some cases. Ceni concludes that these properties reside in certain products of tissue-change which in life are attached to the formed elements of the blood and only after removal of the latter from the body become free in the serum from contact with the "outerworld;" so that in epileptics "a natural auto-immunization is not possible."

4. Storch's comments on Nissl's paper are, as Nissl declares in his reply, "polemical." It is facetious and not of serious value.

PICKETT (Philadelphia).

Monatsschrift für Psychiatrie und Neurologie.

(1902. March, Vol. 11, No. 3.)

1. Further Contribution to the Study of Cerebral Syphilis of the Præcox and Malignant Variety. GIOVANNI MINGAZZINI.
2. The Neuro-fibrillæ in Nerve Cells and Nerve Fibers of the Retina. HEINRICH VOGT.
3. Pathological Anatomy of Paralytic Dementia. THEODORE KÄS.
4. Degeneration of Perception. OTTO GROSS.
5. Experiment of Psycho-physiological Representation of Sense Perception. STORCH.

1. *Further Contribution to the Study of Cerebral Syphilis of the Praecox and Malignant Variety.*—Mingazzini reports 4 cases of cerebral syphilis, one of the præcox variety and 3 of malignant or rapid course. The author believes the autopsy findings show that such cases have fairly constant lesions not found in ordinary syphilis, namely, multiple softenings and hemorrhages in different portions of the brain. From a clinical standpoint the malignancy is materially increased by such infections as scrofula, tuberculosis, malaria and scorbutus. Although most authors hold that the malignant course of syphilis is induced by alcoholic excesses, in one of the author's own cases alcohol and all other secondary infections could be absolutely ruled out. Anti-syphilitic treatment is especially inefficacious in malignant (cerebral) syphilis. Luetics should be warned against the use of alcohol and avoid mental and physical overstrain.

2. *The Neuro-fibrillae in Nerve Cells and Nerve Fibers of the Retina.*—Vogt studied the ganglion cells of the retina in a number of vertebrates, including man, by methylene blue, Holmgren's and Bethe's method. In all his preparations the author found a fibrillary network in the cell, its process an axis cylinder, however the findings were best shown by Bethe's method. The fibers were mostly arranged in definite bundles. The intracellular ramifications of the fibrillæ were exceedingly complex in cells possessing the greatest number of dendrites. In addition to the fibers which passed through the cell to enter a process on the opposite side bundles were often observed broken up at the periphery entrance, some fibers of which entered the adjacent dendrites. Occasionally nerve fibers were found anastomosing with each other without the agency of a cell body (dynamical polaritate). The central cell nucleus was always surrounded by a free non-fibrillary space. Here and there ganglion cells showed bundles which contained fibers arranged rope-like or a large number twining around one or two fibrillæ tendril like. The brain of the calf presents a similar picture in different sections and with different stains. Cell anastomosis was not found in man because of poor specimens. Ganglion cells of the horse showed the best anastomosis. Specimens showed no anastomosis between processes of the same cell. Many axis-cylinders terminated in an actual formation of pericellular network which was in direct continuity with the intracellular network. The author concludes from his extensive studies that a neuro-fibrillary structure of nerve cells and their processes, as well as their continuity, is abundantly proven. Microphotographs and plate drawings accompany the text.

3. *Pathological Anatomy of Paralytic Dementia.*—Abstract to be given at completion of the article.

4. *Degeneration of Perception.*—Gross presents an hypothesis for the explanation of certain mental phenomena seen in the insanities. If the nervous elements show an abnormal tendency to exhaustion the faculty of perception suffers; the individual "fails to grasp the subject" and an intrusion of varied ideas occurs and incoherence results. If added to the foregoing an abnormal excitability remains after exhaustion, a new series of associated perceptions are easily induced, and the clinical picture of mania obtains. On the other hand, melancholia is the result of extreme exhaustion, but with the absence of excitability. A genius shows an absence of fatigue with a lively associative excitability, consequently the sequence of a given series of perception is not broken.

5. *Experiment of Psycho-physiological Representation of Sense Perception.*—Abstract to be given at the completion of the article.

L. P. CLARK (New York).

Allgemeine Zeitschrift f. Psychiatrie.

(1902. April, Vol. 59, No. 1.).

1. Mental Diseases in the Army. EWALD STIER.
2. The Etiology of Mental Diseases Based on Chronic Alcoholism. LUTHER.
3. The Bed-treatment of Chronic Psychoses. ADOLF WÜRTH.
4. Contribution to the Differential Diagnosis of Hysteria and Katatonia. KAISER.
5. The Comparison of the Cerebral Spinal Fluid in Paralytic Dementia and Other Forms of Dementia. SCHAEFER.
6. A Decision of Responsibility. H. KORNFELD.
7. Notes on the Construction of Tropical Asylums. P. C. J. VAN BRERO.

1. *Mental Diseases in the Army.*—In the army statistics there is no tabes or paresis in officers without a luetic history. However, alcohol and trauma precipitate tabes and general paresis very frequently. Hysteria in men is quite common in the armies of middle European countries. In the German army during 1870-71, the traumatic psychoses, which include the epilepsies, was 13 in 100. Army statistics on mental diseases during peace or war are very poor. Better information is hoped for in the future, as an examination on mental diseases is now required of all military physicians on the German army.

2. *The Etiology of Mental Diseases Based on Chronic Alcoholism.*—Luther reports 8 cases of mental diseases developing on a background of delirium tremens. He holds the prognosis in such to be favorable for recovery from the single attack, but relapses are frequent on account of the neuropathic basis generally present. He also reports the histories of 18 cases of alcoholism occurring in the feeble-minded, in which the prognosis was very bad. More or less complete mental degeneration followed rapidly.

3. *The Bed-treatment of Chronic Psychoses.*—As the results of six months' bed treatment of 100 cases of mental diseases the author concludes that the patients in the chronic mania wards were much quieter, and destruction and violence were much less frequent. The necessity of isolation, narcotics and physical restraint was lessened. There were no changes in the body weight of the bed patients. Their general care was made easier and better.

4. *Contribution to the Differential Diagnosis of Hysteria and Katatonia.*—Kaiser holds that katatonic symptoms are often present in hysteria and *vice versa.* The differential diagnosis rests upon a general clinical picture of the two diseases and not upon single symptoms. The essential psychogenic character of both diseases must be borne in mind.

5. *The Comparison of the Cerebral Spinal Fluid in Paralytic Dementia and Other Forms of Dementia.*—Clonic convulsions in epilepsy were materially lessened by lumbar puncture, and the degree of post epileptic coma was also diminished. In dementia paralytica the cerebrospinal fluid pressure was always increased to an average of 182 mm., as compared with 40-100 mm. for the normal. In 2 cases of paresis the pressure fell from 200-270 mm. to 60-80 mm. after paretic convulsions. In another case after inunction treatment of iodides the pressure fell from 200 mm. to 90 mm., and a distinct remission of the disease resulted. In secondary terminal dementia and hydrocephaly and feeblemindedness, increased pressure also existed. A chemical examination of the cerebro-spinal fluid in 12 cases of paresis showed an increased per cent. of albumin, while sugar was diminished; in a lesser degree the same findings held true for the dementias and different states of feeblemindedness.

6. *A Decision of Responsibility.*—Kornfeld presents a translated summary of the expert testimony and court acquittal of a case of paranoia in our own country. The author believes the same court decision would not have been reached in Germany.

7. *Notes on the Construction of Tropical Asylums.*—The author presents a summary of his views in regard to asylum construction in the tropics, with special attention to the economic features. The text is accompanied by a ground plan. L. P. CLARK (New York).

Archives d'électricité médicale.

(1902. February, No. 110.)

1. The Mechanism of the Action of the Electric Arc on the Tissues in Photo-Therapy. H. BORDIER.
2. A New Method of Anesthetizing Teeth by Electricity. RÉGNIER AND DIDSBURY.
3. Technique of Treating Uterine Hemorrhage Employed by Prof. Bergonie. BARIET.
4. The Action of High Frequency Currents (Auto-conduction) upon Animal Thermo-genesis. H. BORDIER and A. LECOMTE.
5. The Present Condition of the Department of Diagnostic and Therapeutic Physics founded by the Hotel Dieu at Toulouse. T. MARIE.
6. Incidence Indicated Adaptable to any Radiographic Couch. H. GUILLEMINOT.
7. An Electro-thermic Pad. H. B.
8. Statistics of the Electro-medical Department in St. André's Hospital at Bordeaux. J. B.

1. *Electric Arc Action.* An experimental research made with the arc light apparatus of Lortet and Genoud to determine whether the erythema produced in the skin from exposure to the arc light is due to heat rays or to chemical rays. In order to apply the heat rays only, the author employed the well known solution of iodine in chloroform, which was circulated through the rock crystal cooler directly applied to the skin. The conclusion is reached that the action of the electric arc light from the above apparatus upon the tissues is wholly due to the

actinic rays of short wave length, and that the erythema or vesication occurring in the tissues under the cooling compressor do not result from heat as one would suppose *a priori*.

2. *Dental Anesthesia.*—A description of a complicated and uncertain method for anesthetizing dental nerves by applying the unipolar discharge from an ondin resonator.

3. *Uterine Hemorrhage.*—Prof. Bergonie employs a strong continuous current (90 m.a.) applied to the uterine cavity by an insulated electrode of special construction, frequently for a quarter of an hour. Data of successfully treated cases are also given.

4. *Animal Thermo-genesis.*—After subjecting rabbits for variable periods of time to electro-magnetic field of a d'Arsonval solenoid traversed by oscillatory currents of high frequency, and then placing them in a d'Arsonval calorimeter, the authors find that under the particular conditions employed by them the heat-producing capacity of rabbits is increased one-thirteenth.

5. Unsuitable for abstract.

6. A description of an adjunct to the X ray tube holder that consists of a metal cross which can be readily and rapidly adjusted to occupy any region of the anti-cathode field.

7. A description of an electro-thermic pad for applying dry heat to any part of the body.

8. Unsuitable for abstraction.

R. H. CUNNINGHAM (N. Y.).

Archives d'électricité médicale.

(1902. March, No. III.)

1. The Radiochronometer and the Experimental Definition of the Different kinds of X rays and similar radiations. L. BENOIST.
2. The Treatment of Facial Neuralgia with Galvanic Currents. VERNAY.
3. The Electrical Installation at the Rennes Medical School. E. CASTEX.
4. The Electrical Treatment of Sciatica. FÉLIX ALLARD.
5. Clinical Facts. Aneurism of the Aorta and Radioscopy.
6. A New Arc Lamp with Iron Electrodes for Photo-therapy. J. B.

1. *Radio Chronometer.*—A description of an instrument for indicating the penetrative power of X rays in which the transparency of a thin disc of silver is contrasted with the relative transparencies of twelve circumferentially-arranged sectors of aluminum of different thicknesses.

2. *Facial Neuralgia.*—In this paper the author relates the history of the successful treatment of fever patients with trifacial neuralgia with strong continuous currents applied daily for from 20 to 32 days. The author summarizes as follows: (1) The electrical treatment of trigeminal neuralgia is a curative one which may be relied upon not only in recent and benign cases, but also in grave cases of long standing if a current of sufficient intensity is employed for a sufficient duration. (2) The current employed should be the constant galvanic. Its intensity should be at least 40 m.a., and may be increased according to the case to 50, 60 or 70 m.a., or even more, and should be applied for from 30 to 60 minutes. (3) The necessary intensity should be used from the first day of treatment if possible, and the applications should continue uninterruptedly for the following 10, 20 or 30 days, according to circum-

stances, even when notable amelioration occurs at the beginning of the treatment. (4) The facial electrode should be at least 150 square centimeters in area, flexible and well moistened. It should be applied over the entire distribution of the trigeminus, from the top of the forehead to the lower jaw, even if the neuralgia is confined to only one branch of the nerve. The indifferent electrode, likewise well moistened, should be at least 200 cm^2 for currents of 40 m.a., or from 300 to 400 cm^2 if stronger currents are employed. (5) The treatment is absolutely harmless if care is taken to very gradually increase the current at the beginning and gradually decrease it at the end of the application, which necessitates a good rheostat, for any sudden variation of the current, although not dangerous, might produce intense vertigo or syncope. (6) Even the strong currents will be well borne if at the beginning the current strength desired to be used throughout the administration is exceeded by 5 or 6 m.a. (7) The active electrode should be preferably the positive; but in case of unsuccess with this pole of a certain number of applications one should not hesitate to employ the negative pole, especially if trophic disturbances are present. (8) The action of the current is probably due to the electrolytic effects produced in the trunks, roots and peripheral terminations of the affected nerve.

3. Descriptive and not fitted for abstracting.

4. *Sciatica.*—After briefly referring to the various causes and kinds of sciatica and sciatic neuritis the author fully reviews the various modes of electrical treatment, such as faradism, galvanization, Franklinization, high frequency currents, etc., preferably to be employed for the treatment of the various varieties of these diseases in a rational manner.

5. *A Description of a Radioscopic Examination of a Patient with Aneurism of the Aorta, accompanied by a good Radiograph.*—The writer concludes that, Radioscopy allows the clinician to rapidly diagnose an aneurism of the thoracic aorta. A tracing made upon tracing paper of the contours of the opacity allows the progress of the disease to be followed. Aneurisms examined with the screen show sometimes feeble pulsations, sometimes strong pulsations. With aneurisms with feeble pulsation no diastolic murmur of aortic insufficiency is found on auscultation, but with those with strong pulsation, usually diastolic murmurs and other signs of aortic insufficiency are present.

6. A description of a small arc lamp containing two iron electrodes instead of the usual carbons, connected in series to give a double arc with the 110 volt circuit. The electrodes are placed in a small hollow metal cup on a handle, with water jacket and removable cap with orifice for the exit of the voluminous ultra-violet radiations which the two iron arcs deliver. R. H. CUNNINGHAM (N. Y.)

Archives d'électricité médicale.

(No. 112. April, 1902.)

1. The History of the Discovery of Electrotonus and the Character of its First Theory. DR. WIASEMSKI.
2. On the Law of the Excitability of Nerves in a Pathological Condition. J. CLUZET.
3. The Electro-medical Equipment at the General Hospital in Tonus. MME. B. MAUREL.
4. Practical Electro-therapy: the Electrical Reactions of the Acoustic Nerve and the Treatment of Some Diseases of the Ear by Electricity. DR. BARRET.
5. Clinical Facts. Dr. Louis Sejuine. A Trial of the Treatment of Cancer with X rays.

1. *Electrotonus*, writes the author, was discovered sixty years ago. Although physiologists in general attribute its discovery to du Bois Reymond, Dr. Wiasemski attempts to show that on the part of du Bois Reymond there was no discovery of the extra-polar currents in 1843, as their existence had been known since 1842, having been recognized and described by Longet and Guérard as "*derivation currents.*" While du Bois Reymond's theory of the nature of the electrotonic currents has been entirely abandoned by physiologists, Lanset's opinion of the nature of the extra polar currents has been developed, although in a modified form, to form the fundamental basis of the theories of Grünhagen and of Hermann. Therefore, to Longet and Guérard the credit of the original discovery should be given.

2. *Law of Nerve Excitability.*—Having shown that Weiss' law holds good for both man and the lower animals, the author contributes a further investigation as to whether or not this law still prevails in frog nerves some days after division. From his results he finds: (1) The formula of Weiss, $Q=a+bt$ is always as correct for the distal portion of the divided nerve as for the normal nerve. (2) If one compares the formulas obtained with the same pole in both cases, the co-efficients *a* and *b* are much smaller for the divided nerve than for the normal nerve. (3) If the formulas obtained with the two poles in both cases, the co-efficients *a* and *b*, which have a minimum value with the active negative electrode on the healthy side, have, on the contrary, a minimum value with the active positive electrode on the side with the divided nerve. After curarization of a nerve where hypo-excitability and inversion of the formular occur, the coefficients *a* and *b* are much greater than in the normal state.

3. Not suitable for abstracting.

4. *Acoustic Nerve Reactions.*—In this contribution to practical electro-therapy the author fully discusses Brenner's, Erb's and Duchenne's methods of using the galvanic and faradic currents in the diagnosis and treatment of ear diseases. The author admits that in normal ears a reaction of the acoustic nerve is not only difficult to obtain but is usually absent even though the intensity of the current is increased to the limit of toleration. When a sonorous reaction is readily obtained with currents of 5 or 6 m.a., where the presence of disease of the external and middle ear can be excluded, hyperemia of the labyrinth or of the acoustic nerve should be suspected. In the treatment of hysterical deafness, tinnitus, and auditory vertigo the continuous or uninterrupted galvanic current, applied according to Erb's method, will frequently produce decided amelioration, or possibly a definite cure of the symptoms. For subacute and chronic middle ear disease faradization is to be preferred.

5. After twenty-nine applications of the X rays to an extensive cancer of the breast, the patient continued to get worse and finally died, although during the treatment the edema and pain in the affected region disappeared.

6. A description of various varieties of electro-medical and X ray apparatus exhibited at a meeting of the physical society.

R. H. CUNNINGHAM (New York).

Revue Neurologique.

(1902. No. 5. March 15.)

1. Results of a Fracture at the Base of the Skull; Apparent Recovery and Death after 17 Years in Dementia and Total Epilepsy. D. ANGLADE and G. CHOCREAUX.

2. Facial Hemiatrophy in Its Relations with Lesions of the Inferior Cervical Ganglion. BOUVEYRON.

1. *Fracture at the Base of the Skull.*—After a very minute report of the case the authors sum up. "First there is the very commonplace accident of a man's falling from his horse and fracturing the base of the skull; he suffered only two months with deafness and facial paralysis on the left side. For over four years he worked as usual, then was suddenly seized with true attacks of generalized epilepsy. The faculties little by little give way, the patient became irritable and impulsive in his dementia, and was confined. At the hospital he behaved like a maniac, incapable of understanding or speaking. Epileptic attacks were almost daily, convulsions generalized. Cachexia extended to all the organs, and the patient succumbed at the age of thirty-eight, seventeen years after the accident, about two after the onset of epilepsy and mental trouble. So much for the clinical facts. The anatomo-pathological statements offer no less interest. How are the symptoms to be accounted for? In the region of fracture, on each side and in the middle part of the third temporo-occipital convolution, there was simply a little line of softening. In the right hemisphere the zones of softening involved the frontal lobe, *influenced it more;* the temporal lobe in its sphenoidal extremity, the inferior third of the two ascending convolutions, and covering these lesions was a very thick dura mater, showing with pachymeningitis. The vessels of the pia matter were obliterated at the circumference of the focus of softening.

What had taken place? Seemingly there was a crack without great displacement of the fragments, hence, facial paralysis and deafness. There was no infection at the time of the accident, but this followed slowly, consequently meningitis, vascular thromboses, softenings and similar symptoms; epilepsy and dementia. Epilepsy might strictly be explained by extended and deep lesion of the frontal lobe; but not if this were partial, since the destructive lesions were unilateral. The microscope explains the fact.

If the macroscopic lesion predominates in front and in the right hemisphere reaches only a little way into the Rolandic zone, inflammation, denoting a violent reaction, begins behind. This was very marked near the paracentral lobule. Perhaps in this diffusion a lesion distinct from inflammatory reactions, and occasioned by some irritant, such as a tumor or abscess, lies the explanation of the epilepsies which occur in patients whose motor zones appear healthy on post-mortem. It must not be forgotten, say the authors, that the present patient showed total epilepsy, which was possibly because the inflammatory reaction passed to the opposite side. This reaction was not alone in the brain. It was found, coincident with important vascular lesions in the protuberance, bulb and in the cervical medulla.

2. *Facial Hemiatrophy.*—In two women, aged respectively thirty-six and twenty-three years, facial hemiatrophy was seen to immediately follow tuberculous lesions of the corresponding apex. The elder, strong and vigorous, with no diseased inheritance or loss or nerve force, but a widow of a phthisical and remarried to a tuberculous patient, became infected with tuberculosis. The lesions were exclusively in the left lung, especially at the apex. Fever and stitch in the side forced her to go bed, and when she arose, about twenty days later, she saw herself that her left cheek was thinner and more hollow than the right. While she had been in bed its skin had seemed "crisp." From that time atrophy of the orbital cavity, face and the greater part of the left side of the neck to the clavicle increased; later it reached over the left side of

the thorax below the breast, encroaching upon the left arm; the patient felt no special pain, neither saw spots nor change of color in the atrophied region. Always cold, she interpreted it as a feeling of shrivelling, or of a mask on her skin. There was no motor paralysis or anesthesia, but perspiration ceased in the atrophied region, little by little, and the skin became much more dry than in the healthy regions on the other side. There was no sensorial trouble. In August, 1899, the symptoms justified a diagnosis of a pleuro-pulmonary lesion, more pleural than pulmonary. The right lung was still normal. While the profile from the right seemed to show the patient as younger, seen from the left she looked sixty. The thin skin was stuck to the adjacent tissues, and it was hard to raise it or fold it. The temple, orbital cavity and the neck on the left as far as the clavicle were entirely atrophied, and this atrophy seemed to reach all the surrounding tissues. On the right the eyelid drooped, although it was not paralyzed, and the reactions of the pupil were normal. The cheek bone, inferior maxilla, masseter and sternomastoid of that side seemed atrophied. Slight atrophy attacked the left arm and thoracic region below the breast. Muscle contracted on faradization, and there was no paralysis of sense or movement, although there were sometimes spontaneous fascicular contractions under the influence of cold in muscles of the face. Perspiration was not secreted on the left side even under most violent exercise or heat influences. Since August, 1899, this hemiatrophy has remained the same, uninfluenced by electric or any other treatment.

The second case was of a young woman whose father had fibrous pulmonary tuberculosis with unilateral chronic sciatica, and whose family history included hysteria and pulmonary bacillosis of congestive form without expectoration. In June, 1899, she was attacked by bacillosis of febrile character, localized at the apex of the left lung. Simultaneously she had neuralgic pain in the left side of face and neck; all nerve endings in these regions were extremely sensitive to touch or to draught, and when thus influenced her skin became very warm and red, the left eye became filled with tears and bloodshot, the left nostril secreted abundantly, and the left side of the mouth secreted a thick and viscous saliva like the white of an egg. At the same time there was cardiac acceleration, pulse increased even from 62 to 133 to the minute. The skin of both hands had become habitually cyanosed and cold, and there was very evident palmar hyperhidrosis; there was marked gastric hyperesthesia, and eating was immediately followed by acute pain. At the beginning of the pleuropulmonary attack, these painful and uninterrupted paroxysms were accompanied by a rise in temperature which varied between 38 deg. C. in the morning, and 39 deg. C. in the evening. At the same time the left apex certainly showed lesions of pleuropulmonary tuberculosis. A new and extraordinary point with this was a very strong breathy sound which extended through the entire track of the left subclavicular artery. There were also very slight but frequent hemotyses.

A fortnight after the last attack the left side of face and neck to the clavicle began to atrophy in comparison with the healthy regions corresponding. For six months this cervicofacial hemiatrophy increased with almost unbearable pain, but at the end of this time the pain seemed to diminish. Repeated revulsion with hot needles was performed on the left apex, hoping to affect the inferior cervical ganglion which the author believed to be the source of all the trouble, and which he supposed was imbedded in a tuberculous pachypleuritic tissue. Nevertheless the therapeutic success was not pronounced enough to affirm with certitude that the sedation of the painful periods was any more the result of in-

tervention than of the natural progress of the affection. At this time the disease seemed to remain stationary. The pleuropulmonary lesions were improved, permanent cold and cyanosis of limbs disappeared and the other symptoms were diminished. Nine months after the onset of hemiatrophy the patient made a pilgrimage to Lourdes and came back free, she said, from all suffering; indeed the psychical improvement was considerable. Moral depression gave way to great hope, and under the influence of the new mental orientation the patient was seized with bulimia, which helped not a little in overcoming the tuberculous lesions. In March, 1900, there was diminution of respiration in the left apex, in the supraspinal fossa as well as in the supraclavicular. The vibrations there were less than in the corresponding side, but there were few râles. The eye remained as before. The temporal fossa and the left cheek had deep hollows, and the skin was adherent to the tissues. The muscles and skin remained as formerly, except for slightly increased dryness. Hair, which in the front parietal region had dried and fallen out while the illness was augmenting, now grew naturally; and most remarkably a frontoparietal hyperstosis, very painful to pressure and like diffuse periostosis, though the patient was free from syphilis, disappeared without specific treatment at the same time that the hair began to grow again.

To interpret these two observations, one must recall, says the author, the situation and branches of the inferior cervical ganglion. This, really a thoracic ganglion, is situated in a small hollow which forms the top of the parietal pleura with which it is in direct relation, and one will see that it may be involved in the processes of pachypleurite of which the apex of the lung may be the source. On the other hand, it is in relation with the articulation of the head of the first side, the first dorsal vertebra, and behind that are the inferior verves of the brachial plexus. Finally, before this in the infraclavicular artery, which Vieussens' valve, when it exists, surrounds completely. Ordinarily it receives the ramifications of the last two cervical verves and of the first dorsal. From this ganglion also radiate the superior fibers which form the vertebral nerve and the anastomose fibers which may be in relation with the carotid plexus through the intervention of the superior cervical ganglion; then the external fibers which follow the vessels of the arm, and finally the internal fibers, to the inferior cardiac nerve or the third cardiac nerve. This distribution and anatomical relation, explain why the patients showed: (1) vasomotor troubles, trophic and secretory of the left half of the face and neck; (2) cold cyanosis and hyperhidosis of arms; (3) crises of cardiac acceleration. The sound of the left subclavicular artery which surrounds Vieussens' valve is explained very well also by a pleuropulmonary infiltration near by. It is judged, then, that the symptoms presented by both patients were the result of a lesion of the inferior cervical ganglion; and it is thought that the lesion was attributable to a process of tuberculous pachypleuritis surrounding this ganglion. JELLIFFE.

MISCELLANY.

EXCISION OF THE SPINE BIFIDA AND ENCEPHALOCELE. John Lithgow (British Medical Journal, January 18, 1902).

Within the past few years the old method of treatment for these affections by injection of Morton's fluid has been almost discarded for the newer and more surgical method of excision. The author reports a case of spina bifida of meningocele variety about the size of a hen's egg. During spasms of whooping cough the tension in the sac became so great that there was danger of rupture, so when the spasmodic

cought subsided the sac was excised. The child was all right a year later. Lithgrow also removed from a child twelve days old an encephalocele as big as the child's head and attached by a pedicle to the occipital region. The child did well after operation, but fourteen days later, when all danger was considered to be past, death suddenly resulted after some convulsive seizures. The tumor was lined throughout with brain substance and was filled with fluid.

W. A. Bastedo (New York).

The Treatment of Toxic Amblyopia. Dr. Terrien (Le Progrès Médical, April 19, 1902).

In toxic amblyopia the treatment is symptomatic after stoppage of tobacco or other poison. For insomnia or nervousness small doses of opium and bromide of potassium may be given for a few days, and then strychnine begun. This is best given in one milligram doses subcutaneously, once a day, but if this treatment is inconvenient it may be given in pills.

℞	Strychnin. sulph.	0.001 (gr. 1-60).
	Quassinæ amorph.	0.01 (gr. 1-6).
	Pulv. rhei.	q. s. ad pil. No. 1.

Take one such three times a day. At the same time electricity, the continuous current in strength just short of being painful, should be employed once a day. One sponge-electrode may be placed on the forehead and the other stroked alternately beneath the eyes.

W. A. Bastedo (New York).....

The Treatment of the Digestive Organs for Mental and Nervous Conditions. Franz Glénard (Le Progrès Médical, March 8, 1902).

The abuse of castration, trephining, sympathectomies, etc., owing to false theories of the causation of disease, has tended to retard truly indicated surgical measures. There is undoubtedly an enteroptosic neurosis, a hepatic neurasthenia and a hepatic agoraphobia, which require for treatment regulation of the diet and laxatives, instead of bromides, hydrotherapy and tonics. In the neuroses, after an alimentary régime, no medication is better than purgatives and chologogues for combatting insomnia, debility, dyspepsia and moral depression. In every neurosis or psychosis the digestive apparatus should be explored, including the liver, intestine, stomach, kidney and spleen as to their size, consistency, sensitiveness, location, relation and degree of fixity. The organ most frequently at the bottom of a neurasthenia is the liver, and who would deny that the mental functions become sluggish when the digestive tract is working poorly. Macpherson, at the Stirling insane asylum, submits all the patients to lavage of the stomach, laxatives, intestinal antisepsis and strict diet. Glénard cites several cases, as follows: A young woman with enteroptosis who had eaten some forbidden food, wept and became highly neurotic, but soon after the administration of sodium bicarbonate became quite herself again. A young man of sedentary habits was attacked with neurasthenic anxiety and digestive cachexia with vomiting, so that it was feared that he would die. He recognized nobody and talked as if insane, but after vomiting excessively following tickling of the uvulva, he became rational, slept well, and the next morning was in his usual health. Therefore there do exist in digestive pathology conditions in which the effect on the nervous system is undeniable. Piqué says that digestive intervention is especially efficacious in the psychoses of depression, as melancholia. The author had such a case with gastro-hepatic crises, in which stenosis of the hepatic flexure of the colon was

made. An operation found the colon in this region tightly bound by adhesions, and left them broken up; now there are no more psychical manifestations. The author believes that in psychic states as well as in neurasthenia a cure results not from direct treatment of the nervous system, but from medical or surgical treatment of one or other of the viscera. W. A. Bastedo.

Treatment of Insanity by Continuous Baths. P. Keraval (Le Progrès Médical, May 3, 1902).

The baths at Heidelberg used by Kraepelin and W. Alter are continued day and night for weeks and months, the patients eating from tables placed over the bath, and being allowed to smoke, read, crochet, etc. At night a rubber air-pillow is supported beneath the head. The water is kept at 34 deg. C. (93 deg. F.)

Such baths are specific in mania, agitation and the acute delirium of general paralysis. By suspension in water in a hammock the feeble and paralytic escape injuries and bed-sores. Almost without exception the patients accept the treatment, though in some cases it must be begun in short seances. The ordinary functions of urination and defacation make the most trouble, but not more than they do in a cell. The patient is quickly calmed and rapidly gains an appetite. It is not of much use in epilepsy, but for delirium tremens it is valuable, combined with caffeine in large doses. Alzheimer reports success with these baths at Frankfort, twelve bathers being constantly employed. Bieberbach has used them for a year at Heppenheim with very favorable results. In periodical insanity the subsequent attacks are especially mild. Insomnia is never persistent. W. A. Bastedo (New York).

Infantile Convulsions. Prof. Déspine (La Méd. Moderne, April 9, 1902).

Infantile convulsions may be divided into two main groups, symptomatic convulsions, due to disease of the cerebral nervous system (meningo-encephalitis, tumors, hemorrhages, etc.), and idiopathic or essential. The latter group includes three main forms, external convulsions(eclampsia), internal convulsions (spasm of the glottis), and contractions of the extremities (tetany). The idiopathic forms are here studied as to etiology and symptomatology. Heredity plays an important part in causation. The influence of rickets is that of an autointoxication. Dentition is of no importance. In the newborn convulsions are usually due to organic lesion, and the prognosis is grave. Often asphyxia is a cause, and is due to congenital cyanosis, whooping-cough, bronchopneumonia. Many cases are attributed to poisoning by lead, alcohol, opium, santonin, etc., The most frequent cause is autointoxication (uremia, athyroidism, hypertrophy of the thymus, gastro-intestinal disease). Reflex eclampsia may be due to irritation of the skin, ears or digestive tract. Clinically, eclampsia in infants is preceded by a stage of hyperexcitability of the nervous centers, manifested by hyperesthesia, exaggerated reflexes, electrical hyperexcitability. As to form, the convulsion is neither purely tonic nor clonic; exceptionally a purely tonic convulsion may occur. Commonly the spasm begins at the eyeball, extending to the face, neck, extremities and trunk; if localized, the face is usually the part involved. Bilateral symmetry is the rule; exceptionally the spasm is exaggerated on one side. Spasm of the glottis is characterized by a series of inspiratory or expiratory sounds; the paroxysm may end fatally by syncope, but typical cases are described which were saved by tracheotomy or by interbation. Spasm of the glottis is characteristically a disease of the first year of life;

more than 50 per cent. of all cases occur during the first six months. Compared with general convulsions or with spasm of the glottis, tetany is a rare condition. In 10,000 infants, tetany occurs 6 times; spasm of the glottis 31 times; eclampsia 61 times. JELLIFFE.

THROMBOSIS OF THE CAVERNOUS SINUS; REPORT OF FOUR CASES, WITH ONE CRANIAL OPERATION. E. W. Dwight and H. H. Germain (Boston Med. & Surg. Jour., May 1, 1902).

The diagnosis of this condition has rarely been made. The literature includes 178 cases in addition to 4 cases reported by the authors. Most authors agree that the treatment is entirely preventive. Those who suggest operation usually speak only of drainage through the orbit. In the case operated on by Dwight the sinus was revealed by trephining the temporal bone; this same operation was performed a month later in a case described by Hartley and Knapp. These two cases are held to justify the belief that thrombosis of the sinus is distinctly an operable condition. Incision into one sinus apparently relieved instantly and completely the interference with circulation in both. The cranial operation is not associated with extreme difficulty; it can be done under almost primary anesthesia; it is not associated with any degree of shock and it can be completed in a few minutes; the hemorrhage can be easily controlled. JELLIFFE.

SOME POINTS ON THE MANAGEMENT OF THE NEURASTHENIC. James H. McBride, M.D. (Journal of the American Medical Association).

Overwork alone rarely causes nervous breakdown. Worry and anxiety, prolonged mental application without rest are frequent causes; underlying which is an unborn instability of nerve element and derangement of the alimentary tract. The mental element in neurasthenia is important. It is shown by inability to fix the attention, the feeling of mental tire, loss of interest, the weakening of volition, the impairment of memory. The thoughts are always fixed on the body, and are limited to a set of morbid sensations, such hyperconsciousness of bodily ailments becoming often a true hypochondria.

The writer advises partial rest, rising at 9 o'clock, and resting two hours in the middle of the day, or complete rest if there is debility; but rest cures should not be too prolonged.

The personality, good judgment and discriminating sympathy of a trained nurse are all important. Frequent feeding, milk diet are not applicable to all cases. Exercise is essential and yet can do harm. Neurasthenics frequently have exaggeration of the feeling of tire and fatigue. If they have "anesthesia of the sense of tire" they will overdo without knowing it. Outdoor walks should be taken on the ground, not on pavements or verandas. Hill climbing should be reserved for convalescence. Morning cold baths are injurious if much below 78 deg. Mental symptoms need mental treatment.

W. B. NOYES (New York)

CAISSON DISEASE. A. H. Muir Macmorran (Brit. Med. Jour., April 26, 1902).

A partial recognition of some of the causes of this interesting modern malady has done much to decrease its frequency. The author cites a series of cases which developed in the recent building of the Greenwich footway tunnel. One of the chief reasons why there is less caisson disease than formerly is that in most instances the men are now subjected to a very careful and searching medical examination. The total percentage of rejections (made by the writer and his colleagues) of appli-

cants for permission to work underground was 18.8. The most important grounds for rejection are a high-tension pulse, booming heart sounds or reduflication of the second sound. This supervision is the first means of combating caisson disease. The second consists in reducing the CO_2 as far as possible. This was accomplished by the introduction of a blow-off tube in the shield and by injecting the air through screens of caustic soda. Macmorran believes the theory of hyperemia has been set aside by many on altogether insufficient grounds. He thinks that the true explanation of caisson disease lies in the combining of the two factors most frequently discussed in connection with it, namely, hyperemia of the deeper tissues and accumulated impurities in the blood, due to imperfect interchange of gases in the lungs. Nerve sedativs are to be preferred to opiates in the treatment of caisson disease, and in severe cases cannabis indica often gives excellent results. The nitrates and acetates of potassium are useful in the termination of the trouble, but by far the most important part of the treatment is the medical lock into which air passed over caustic soda is pumped till the pain subsides. Caisson disease will become a thing of the past if only suitable men are employed and if the suggested treatment is given to the injected air.

JELLIFFE.

LESIONS OF THE CONUS MEDULLARIS AND CAUDA EQUINA. Bertram W. Suppy, M.D. (Journal of the American Medical Association, May 10, 1902).

The conus medullaris occupies a position in the spinal canal directly behind the first lumbar vertebra, completely hidden from view by a mass of firm and coarse fibers, constituting the beginning of the cauda equina. Recent writers limit to that portion of the cord represented by the third, fourth and fifth sacral and coccygeal segments. The clinical picture produced by lesions of the conus medullaris is characterized by impairment of sensation over an area which involves the integument of the penis, scrotum, perineum, anus, inner aspect of the buttocks and posterior surface of the thighs. The sensibility of the mucous membrane of the penis and rectum may also be dulled. If the lesion is sufficiently destructive, loss of muscular power of the bladder and rectum may be seriously impaired, sexual power lost and bed sores may develop. When disease of the cauda equina is accompanied by typical symptoms it is easily recognized. Except when due to trauma, disease of the cauda usually develops slowly, producing symptoms more or less characteristic of root disease. The patient first experiences pain upon movement of the lower extremities; later the pain becomes spontaneous and persistent, with exacerbations. Subsequently anesthesia begins, and when the lesion is a uniform compression of the cauda, the function of the central fibers may be first disturbed. Bladder and rectum symptoms may appear early. Muscular weakness is present in proportion to the pressure on the motor fibers. There may be exaggerated reflexes at the beginning; later they are diminished and finally lost. Atrophies develop, electrical reactions may be altered. That which specially distinguishes diseases of the cauda equina is pain. A conus lesion may be associated with pain, if the cauda or meninges are involved; otherwise there is absence of pain. Differential diagnosis is important as disease of the cauda equina may often be amenable to surgical treatment.

W. B. NOYES (New York).

EPILEPSY, ITS ETIOLOGY, PATHOLOGY AND TREATMENT BRIEFLY CONSIDERED. William P. Spratling, M.D. (Journal of American Medical Association, May 3, 1902).

Epilepsy may be classified according to three epochs of life: (1) up to the age of 20 years; (2) during adult life; (3) after the beginning of senility. More than three-fourths of the cases occur in the first period. The epilepsies of the first period may be grouped under two heads: primary, or developmental, and accidental. The latter also occurs during the adult period, but with far less frequency. Two kinds of heredity must be considered: similar and dissimilar, the former meaning the transmission of the same disease from parent to child, the latter meaning that alcoholism, insanity, tuberculosis, chorea or a kindred affection in the parent is changed in type in transmission so as to appear as epilepsy in the child. In a study of 1,100 cases of epilepsy the author finds 16 per cent. due to similar heredity, 40 per cent. to dissimilar heredity, such as alcoholism in the ancestors, 16 per cent. in the males, 12 per cent. in the females; insanity in the ancestors 7 per cent., tuberculosis 14 per cent. Of accidental forms 11 per cent. were cerebral palsies; primary or developmental epilepsy after 20 years is probably never met with. After this age it is due to syphilis or traumatism, to alcoholism, ptomain or chemical poison. The writer believes that the sensory or angular cells in the second layer of the brain exercise a power of control over the motor cells in the layer above. When these sensory cells become weakened or destroyed they lose their power of control, and the motor cells explode; that is, they suddenly and violently give off their force at irregular intervals. The structural incompleteness of the sensory cells and consequent failure of inhibition, rather than the over production of nerve force by the large motor cells, is responsible for these periodic discharges.

The writer notes three specific lesions after paroxysmal discharges in uncomplicated epilepsy: (1) a partial disappearance of the tangential fibers of the cortex, "the neurones of association," so-called, and similar to lesions described in cases of general paralysis; (2) an infiltration of nerve cells by leucocytes; (3) the disappearance of the fine granules in the protoplasm of the cell substance of the cortical nerve cells. The latter are probably due to chemical changes dependent on nutritive processes. In treatment the use of bromipin, a brominized oil of sesamum, is advocated. W. B. Noyes (New York).

Pseudomeningitis. T. Donath (Deut. med. Woch., April 17, 1902).

The presence of a symptom-complex resembling meningitis, without the demonstrable lesions of this disease at a later autopsy, has sometimes been noted, especially during epidemics of influenza. An unusual case of this kind is reported. The patient, a young man of eighteen, developed eleven days after an osteotomy for genu valgum a sudden rise of temperature, severe cerebral symptoms, loss of consciousness, muscular spasm, retracted abdomen, etc., with death following on the third day. Fat embolus and uremia could be properly excluded, and all the signs pointed to meningitis, possibly tuberculous. Autopsy showed some edema of the pia and surface of the brain, but no signs of any acute inflammatory changes. The inufluenza bacillus could be readily demonstrated in some of the lobules. The author calls attention to the diagnostic value of lumbar puncture in these cases, when ordinary means are of doubtful value. Jelliffe.

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Original Articles.

POLIOMYELITIS OF THE ADULT.

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The occurrence of a true poliomyelitis in the adult, with acute onset under conditions suggesting an infection, is no longer disputed. Such cases have been reported by Gombault[1], Bernhardt[2], Cornil and Lépine[3], Williamson[4], Grawitz[5], Niedner[6], Jagiç[7], Hoppe[8], Caverly[9], and others. The confusion regarding the relationship of this affection to others, notably acute ascending paralysis (Landry) appears to be increasing rather than diminishing. The following report is made because relatively few cases with autopsy have been published, and also in the hope that the matter of classification in view of our growing knowledge of the pathological anatomy is of sufficient interest to permit a reopening of the general subject.

Case I.—J. C. L.,* an American by birth, unmarried, twenty-five years old, worked chiefly indoors, lifting. He had been especially overtaxed just before his present illness, but had not

*For the following clinical history I am indebted to Dr. E. M. Buckingham, Boston City Hospital, Dr. J. J. Minot, Carney Hospital, and Dr. Pillsbury, formerly a student of medicine at the Harvard Medical School.

been exposed to cold or similar influences. His family history was good, and his personal history showed no undue excesses in tobacco, alcohol or stimulants of any sort. He had had gonorrhea six years before this illness, followed three or four years later by a slight stricture. He had also had soft chancres, but sufficient evidence of syphilis was lacking. He had not suffered from rheumatism. His present illness began September 11, 1898. The onset was, to him, similar to previous experiences in taking cold. There was headache, backache, pain in the extremities, without sense of fever or chilliness. During that day he grew worse and he had a night of poor sleep. On the following day (Monday), he felt no better and could eat little. Thinking he had "grippe" he started to "walk it off" and kept on his feet the greater part of the day. His legs felt as well as they ever had, but his back was weak and gave him considerable pain when he suffered a jar, as in stepping off a curbing. He ate no dinner, but took a drink of whiskey, went to bed early and slept well. The next morning (Tuesday), he rose early to get whiskey, but immediately fell; he pulled himself to his feet by the aid of the bedpost, but found that he could not stand. He was, however, able to creep. He got his drink and went back to bed. He then found that he could not move his feet or toes. He had some nausea and vomited. A physician was called, who found the temperature to be 103.5°. He was forthwith sent to the Boston City Hospital in the service of Dr. E. M. Buckingham.

The following day (Wednesday), both thighs were paralyzed; he could not sit up nor turn in bed. The next day his arms were weak, particularly the right, and he was unable to breathe deeply, except by grasping the head of the bed and thereby bringing the accessory muscles into play. Sensation, he thought, was at no time lost in any part of his body, but there was at first a slight feeling of numbness in the limbs; they felt alternately warm and cold, and were often covered with sweat. This was especially noticeable on the right leg. The paralysis was flaccid from the first.

For the first seven days after entrance, the bowels moved only by enema; the catheter was used for two days; after that there was no difficulty with bowels or bladder. The temperature remained elevated for four days, became normal two days later, and remained so. The special senses were at no time disturbed; the appetite in general was good, and sleep was satisfactory. For about a week after entrance, there was some pain in arms and legs. His back ached when moved, otherwise not. The muscles of face, neck, deglutition and mastication were not affected.

About a week after entrance to the hospital and about ten days after the onset of the disease, voluntary movements of the arms became stronger, and the breathing less labored. A week later he began to regain slight control of his toes and feet and soon after of his hips. After November 1 he began to sit up an hour a day, supporting himself in an erect position by aid of his arms. His back remained very weak.

The physical examination on entrance showed no Widal reaction and no evidence of plasmodia in the blood. The urine had a specific gravity of 1023, without albumin or sugar, or anything significant in the sediment. November 9, pupils and ocular muscles were normal. The teeth were in good condition and showed no evidence of metallic poisoning. Cranial nerves were in general free of involvement. The chest was thin, movements feeble, but equal on the two sides; the accessory muscles were not used. Inspiration was feeble and prolonged; respiration was 28 and regular. Palpation, auscultation and percussion showed nothing abnormal. The heart was normal with a somewhat rapid pulse. The abdominal organs were negative. The arms were thin and the muscles flabby, the skin warm, moist and of natural appearance. Voluntary movements, both of flexion and extension, could be weakly made. The left hand-grasp was particularly weak. The legs were thinner and the muscles more flabby than in the arms; the skin was normal. Voluntary movements at the hips very weak; the flexors of the legs could be seen to contract, but not with sufficient strength to induce the slightest flexion of the knees. Motion of the feet and toes was possible, but weak. Passive motion was not limited in any joint. The knee-jerks were absent.

Sensation for contact, pain and temperature was not impaired. There was tenderness on either side of the vertebral column in the region opposite the angles of the scapulæ, and in the axillæ and popliteal spaces, without marked muscular tenderness. No notes of an electrical examination are obtainable.

November 11, the patient contracted a cold, during which he had difficulty in expectoration and respiration; his cough was feeble; the breathing was vesicular, with numerous râles. No tubercle bacilli were found in the sputum. November 14, the cough had diminished, respiration was easier and the lung condition decidedly improved. Slight improvement in movements also occurred and a very slight knee-jerk was obtained. There was no sign of contracture, beyond a tendency to keep the knees flexed.

By his own request the patient was discharged from the

City Hospital, and admitted to the Carney Hospital in the service of Dr. J. J. Minot.

November 22, examination showed essentially what has already been described. The knee-jerk was reported as again lost, with very slight plantar and cremasteric reflexes. Atrophy of legs and thighs was noted, and very limited movements. A fundus examination showed nothing abnormal. The patient was hopeful, and thought the movements of the legs were slowly improving. A certain amount of flexion at the right knee was possible, as well as movements of the feet and toes.

December 7 there was complaint of dyspnea which increased, though apparently not to an alarming degree. At 4 A.M., December 8, he appeared to be comfortable; when next seen an hour and a half later he was dead. His temperature had remained normal during his stay in the hospital.

This case presents a typical picture of poliomyelitis as usually met with in the adult; unexplained ill feelings with rise of temperature, followed by paralysis, involving all four extremities, but predominantly the legs, which reaches its height in one to three days, and then tends towards improvement; some subjective disturbance of sensation, passing in a few days, leaving a condition essentially of motor paralysis of the muscles of the trunk and extremities, without at any time disturbance of consciousness.

Autopsy—By Dr. G. B. Magrath, Dec. 8, 1898; six to seven hours after death. Other organs than the central nervous system showed nothing of importance. The brain, cord and a portion of the right sciatic nerve were preserved for microscopic examination. Portions of the brain—paracentral lobules—and of the oblongata and cord were hardened in alcohol for later Nissl staining. The remainder of the cord was preserved in 10 per cent. formalin, and Müller's fluid, the sciatic nerve, in Müller's fluid, and the brain in 5-10 per cent. formalin. The brain was normal in external appearance, except for slight adhesions between the dura and pia at the tip of both temporal lobes, but much more marked on the left. The cord also was normal externally, but on section at various levels, there was a very striking injection of the ventral horns, throughout the cord, marking them out sharply from the surrounding white matter. A culture made from the subdural space of the cervical region gave no bacterial growth.

Microscopic Examination—A study of the cord at var-

ious levels shows throughout its entire extent an almost complete distintegration of the ventral gray matter, extending to a considerable extent into the dorsal horns, and slightly into the surrounding white matter, confirming the appearances seen macroscopically. A more detailed study of the cord from below upward gives the following appearances: A section through the conus terminalis shows an exceedingly slight accumulation of cells in the neighborhood of blood vessels; otherwise there is at this level no abnormality. In the sacral

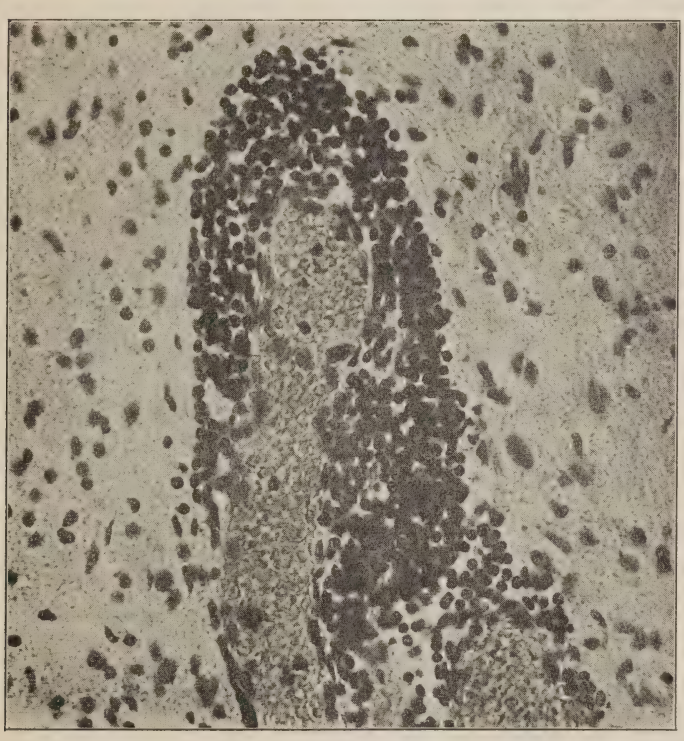

Fig. 1. Infiltration of a perivascular space with extravasated cells.

cord and lower portion of the lumbar enlargement the evidences of inflammation become more apparent. The perivascular spaces are in many areas infiltrated with extravasated cells, both in the ventral and dorsal horns. A Weigert section shows a clearly marked, though not complete loss of myelinated fibers in the ventral horns, loss of many nerve cells and partial degen-

eration of ventral roots. The blood vessels throughout the section, notably in the pia and nerve roots are completely distended with blood, but nowhere except in the ventral horns and portions of the dorsal horns does there appear to have been an exudate from the vessels. The cross-section of dorsal nerve roots at this level shows in several places small areas of necrosis which have the appearance of dependence upon local vascular disturbances.

At a slightly higher level in the lumbar enlargement the pathological process is very much more extensive, and the appear-

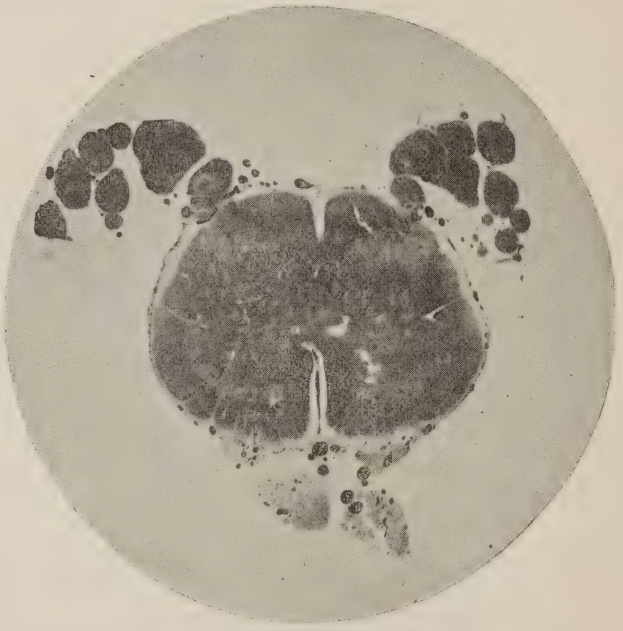

Fig. 2. Section stained by the Weigert method, showing partial degeneration of ventral roots.

ances here will serve as a type of the character of the alterations throughout the cord. A hematoxylin-eosin specimen shows the end results of a violent inflammation. The same conditions, in general, prevail as those already described, but much greater in degree. The ventral horns are filled with distended capillaries in the neighborhood of which are many extravasated cells. It is noticeable that among the cells of this exudate no leucocytes are to be seen, which is not remarkable in consideration of the fact that three months had elapsed from the acute onset

of the disease to the death of the patient. The nuclei of the remaining cells are for the most part small, round, deeply staining with hematoxylin; in general of the lymphoid variety. Occasional, elongated, somewhat vesicular nuclei may be seen; it is possible that many, or perhaps all of these nuclei may belong to proliferating neuroglia cells, as part of the reparative process; no mitoses, however, are discoverable. The general dilatation of capillaries extends distinctly into the dorsal horns and to a less degree into the white matter and vessels of the pia. The lesion is, however, so much more marked in the ventral horns, that that may be regarded as its essential location.

Sections stained by the Nissl method are of particular interest. The appearances in the lumbar region may again serve as a type of the changes found throughout the cord. Most noteworthy is the diminution in the number of nerve cells. At one of the levels studied in the lumbar enlargement not a single large ventral horn cell is to be seen on one side, while on the other the number is markedly reduced. The cells destroyed by the original inflammatory process have for the most part entirely disappeared, while the relatively few remaining cells are frequently normal as regards their markings, a condition to be expected in a local traumatic process as poliomyelitis may properly be regarded, so far as the nerve cells are concerned. Occasional cells may be seen, showing marked chromatolysis, and distinct dislocation of the nucleus, having the general appearance of cells undergoing the so-called degeneration at a distance, a type of degeneration which might easily occur in a destructive process of this sort. It is often difficult to trace certain processes to a distance from the cell body, and in some instances the dendrites appear as if broken, probably by the violence of the original exudation. There is no sufficient evidence of cell disintegration due to a slowly acting toxic cause. The point of chief interest in the appearances given by the Nissl method is the comparative lack of transitional forms between a complete destruction of nerve cells and wholly uninjured cells.

There is no distinct evidence of the process described by Marinesco as restitution of injured cells, possibly owing to the insufficient time which had elapsed since the beginning of the illness. Swollen cells with abnormally deeply staining chromatin are not visible, although many cells show an unusually definite coloration of the Nissl bodies.

The changes described for the lumbar region extend throughout the cord with essentially unvarying intensity, and therefore require no special comment. In the cervical region a few large cells remain intact and a considerable number of smaller cells, faintly staining and much degenerated, may be

seen on careful search with a high power. The vascular changes are as pronounced here as at other portions of the cord.

Slightly higher, in the lower oblongata, the vascular distention and exudation have almost wholly disappeared. The mid-oblongata at the level of the hypoglossus nucleus shows no abnormality. The cells are well stained and sufficient in number. The upper oblongata, pons, cerebellum and cerebral cortex are normal, beyond an occasional extreme distension of blood vessels, to which no special pathological significance is to be attached. There is nowhere evidence of exudation from the vessels. Beyond the distension of the vessels, especially marked in the cord, the walls show no abnormality. The sciatic nerve is very markedly degenerated. Certain of the bundles have almost completely lost their myeline (Weigert); whereas others remain essentially intact. This observation is of some value as showing the relative arrangement of motor and sensory nerves in the peripheral trunks. It is also of interest that sections stained by Marchi's method show no degeneration of the myeline, probably due to the fact that three months had elapsed since the onset of the disease.

In summarizing the chief alterations observed the following points are to be noted: Essential limitation of the pathological process to the spinal cord; in the cord, chief though not exclusive localization in the ventral horns; evidence of primary inflammation with secondary degeneration and destruction of nerve cells; marked degeneration of peripheral nerves.

The clinical course and pathological anatomy of this case correspond closely to several others which have been reported within the last few years, and to which allusion will later be made.

Case II.—A girl of seventeen, up to that time well and athletic, was taken ill with vague symptoms, referable to her head with some pain about the body; she complained of dizziness and general weakness. This condition persisted until she was forced to take to her bed at the end of about three weeks, with an acute febrile attack, the temperature at one time reaching 103.8. During this acute attack, or immediately following it, a rapid loss of power in the arms and later in the legs developed, which became practically a total paralysis, accompanied by a somewhat sighing respiration.

The patient vomited easily and often; there was retention of urine and feces, necessitating the use of the catheter and en-

emata. There was also temporarily excessive pain in the legs, of a lancinating character.

Physical examination made soon after the onset of the paralysis showed complete motor disability in both legs and in the right arm. The left arm was also practically helpless, except for some flexion at the elbow and wrist; extension was impossible. The cranial nerves were not involved, nor was the phrenic to a noticeable degree. Sensation was uninvolved, except for a possible slight hyperesthesia of the legs; there were no pains as at first. The deep reflexes were lost; the temperature had returned to normal and the general condition was much improved over what it had been. The urinary and rectal disturbance, however, persisted.

At a second examination, made about a month later, this loss of control of the sphincters had passed; it had not been necessary to use the catheter after the first week of the onset of paralysis. There was in other respects also, some improvement; more motion of the left arm was possible, but it was still very weak. The right arm was slightly movable at the shoulder joint only. The right leg remained absolutely paralytic; likewise the left leg, except for slight movement of the toes. The muscles were flabby and showed evidence of atrophy. Sensation remained essentially unimpaired, and the sharp pain of the earlier stage had entirely disappeared. Respiration was now unaffected, and the mental state was clear.

The patient later, fortunately, came under the treatment of Drs. E. H. Bradford and E. G. Brackett, of Boston, who through assiduous training of the muscles, over a long period of time, were able to bring about a degree of improvement which it is not to be supposed that nature unaided could have done. Nearly two years after the onset of the disease, Dr. Bradford writes that the patient is able to stand and take a few steps with the help of crutches, and that progress had been made during the last six months.

The following case, occurring in a boy shortly before puberty, is of importance as illustrating a cerebral complication or accompaniment of the disease which is not altogether easy of explanation, except through association with an accompanying encephalitis.

Case III—N. A., a boy, fourteen years old, came to the Nerve O. P. D. Massachusetts General Hospital (No. 22,174), March 31, 1902, with the following history: Toward the end of August, 1900, after a bicycle ride of about fourteen miles,

not associated with special fatigue, and after wading a few hours later, he felt chilly; he was not ill that night, but the next day had severe backache and headache. The following day he had considerable pain in the right foot. On waking the next morning, after a good night's sleep, his right leg was paralyzed. That afternoon the left leg began to grow weak and the next morning it also was paralyzed. On this day fever was first noticed, though it had probably existed for several days, and he began to grow somnolent. He rapidly sank into a stuporous condition from which he could not be roused by any ordinary stimuli, and remained so for about eight weeks. He did not speak during this time, except on one occasion, about two weeks after the onset, when he is said to have conversed intelligently for about half an hour. After that he became more stuporous with increased fever. His bowels and bladder acted involuntarily, but he was always able to swallow. There was at no time paralysis of any of the cranial nerves; the father gives an extremely doubtful history of retraction of the head. Recovery from this condition was gradual, with increasingly prolonged intervals of consciousness. The patient remembers absolutely nothing of this period of eight weeks; his mental state is now entirely normal. He had had no suppuration of the ears; and beyond chicken-pox the previous spring and headaches with constipation, he had previously been well. An analysis of the drinking water showed the prescence of lead, and a slight amount was also found in the urine.

After recovering from the stuporous condition spoken of above, all four extremities were more or less paralyzed, the legs to such an extent that he has not since been able to stand or walk, and the arms to a less degree. There has been no disorder of the sphincters nor of sensation.

Physical examination at the present time shows the following conditions in detail: General appearance of health: the patient is well grown and of good color. The head and cranial nerves show no abnormality whatever. The pupils are large, equal and respond to light. The fundus shows nothing pathological. The patient has a rachitic chest. All the muscles are soft and many show distinct atrophy. The trapezii and sternomastoids are not involved. Movements of the shoulders are somewhat weak; the muscles of the upper arms are flabby, but individual motions are preserved. The right arm shows greater atrophy than the left. In both arms there is distinct atrophy of forearm muscles on the ulnar flexor side. The hands are similarly atrophic, but more marked on the right. The small muscles of the thumb are particularly involved, permitting but very slight movement on the right. The interossei are some-

what affected. Extension of hands at the wrist is possible. In respiration the movements of the ribs are imperfect. There is some lordosis. Right glutei completely paralyzed; left approximately normal. The legs are much more atrophic than the arms, practically all the muscles taking part. The patient as stated, cannot stand, but creeps with comparative ease, flexing the legs both at hips and knees. In the sitting posture he cannot raise either leg at the hip nor extend the leg on the thigh. If knees are supported, flexion and extension are slightly possible. Abduction and adduction at the thighs imperfect, particularly on the right. All movements of the right foot are abolished; extension of toes of the left foot possible; flexion lost. Slight flexion and extension of the ankle possible. The feet are cold; the right foot somewhat cyanotic.

Reflexes, superficial and deep, are lost; the paralysis is flaccid; there are no contractures.

Electrical examination shows quantitative diminution to both faradism and galvanism, without qualitative change in the muscles examined.

During the past four years and three months at the Out-Patient Neurological Department of the Massachusetts General Hospital, anterior poliomyelitis has been diagnosticated seventy-six times. Of this number ten occurred in persons upward of twelve years of age; five of these eighteen or more years old. In several of these cases, however, the diagnosis permits of doubt, so that, judging from these figures, the percentage of adult cases, uncomplicated by cerebral symptoms, is very small.

Case IV—L. F., twenty-two, unmarried, a school teacher, at nineteen was taken with an illness supposed to be influenza. On the third day of this illness a sudden paralysis of the legs came on which had prohibited walking since. There was no sensory nor sphincter disturbance; there remained practically a complete paralysis of the right leg, and a very marked paresis of the left. There was double toe drop; the skin of the feet was cold and brawny in appearance. Probably much muscular atrophy, but masked by fat. The knee-jerks were absent.

A typical case of poliencephalitis associated with a cervical poliomyelitis occurred during this period in which the cerebral symptoms so far predominated that it is rather to be considered in another category than poliomyelitis.*

*This case will be alluded to later in this paper and reported in detail in another place. See in this connection, Sherman and Spiller: "A Case of Poliencephalomyelitis in an Adult." Phil. Med. Jour. v. 734, 1900.

Symptomatology.—The cases described above conform to the generally accepted picture of acute poliomyelitis. Had they occurred in infants or young children, no doubt whatever would have been entertained of the diagnosis. A febrile attack, with general constitutional symptoms, followed by a more or less extensive paralysis of one or more extremities of a purely motor type, rapid in onset, forms a characteristic picture at whatever age it occurs. In spite of an increasing number of clinical and some pathological observations showing conclusively the existence of acute poliomyelitis in the adult there still remains a certain prejudice against the acceptance of the diagnosis, so deeply rooted is the feeling that it is peculiarly a disease of childhood. It is unquestionably a rare affection after the fifteenth year, but no one who has studied the reported cases can for a moment deny that it occurs in characteristic form, and probably with much greater frequency than ordinarily supposed. Bernhardt[11] in 1874 reported three cases in men, two of which involved all four extremities. He notes the frequency of this extensive involvement in the adult, an observation made and confirmed by others since. In 1875, Cornil and Lépine[11] reported with autopsy a case of relatively slow onset, with characteristic cord changes, which others might have been inclined to regard as a case of so-called Landry's paralysis. Williamson's[12] case was a man of twenty-two, who died five weeks after a sudden paralysis of both arms and legs, without permanent sensory disorder, and in which the autopsy confirmed the diagnosis. Grawitz[13] narrates the case of a woman of eighteen, with involvement of all four extremities, followed by partial recovery, the paralysis in this case being of somewhat slow onset, occupying several days to reach its maximum. A somewhat similar case in a man of nineteen, followed by death with paralysis of respiration is reported by Niedner[14], without autopsy. Jagiç[15] observed a case of primary involvement of the leg, extending to one arm, followed by death in which the similarity to Landry's symptom-complex is noted. Hoppe's[16] cases, although not fatal in outcome, admit of no reasonable doubt in diagnosis, and Caverly's[17] recurring in the famous Rutland, Vermont, epidemic of 1894, are equally beyond suspicion, although no autopsies were obtained. Of special inter-

est on the clinical side are those cases which somewhat arbitrarily have been classed as Landry's paralysis, at times in spite of lesions found post-mortem which would naturally place them in the category of anterior poliomyelitis. Among these are the observations of Bailey and Ewing[18], J. J. Thomas[19] and Greene, Wilson and Rothrock[20], all of whom describe lesions (see remarks on pathological anatomy) essentially characteristic of poliomyelitis, and yet classify their cases under the vague heading of Landry's paralysis. The tendency to consider cases of rapidly advancing paralysis, whether fatal or not, and regardless of the pathological findings as Landry's paralysis is very noticeable in much of the best recent work in America. An attempt to fit pathological changes into a preconceived clinical picture usually leads to an unnecessary confusion, and such has been the result here. We have elsewhere[21] discussed what to us seems the proper attitude to assume toward the complex of symptoms which goes by the name of Landry. We wish, now, merely to reiterate that the clinical course of the disease must not be the sole criterion of its nature. Poliomyelitis is none the less poliomyelitis because it simulates in its clinical course what Landry many years ago described as an acute ascending spinal paralysis. German observers have on the other hand been quick to recognize the occurrence of a form of poliomyelitis in the adult, in which the paralysis has been of relatively gradual onset and often ascending in type. In 1876 Schultze[22] reported a case of poliomyelitis with autopsy in which there was a resemblance to a rapid Landry's paralysis. Again in 1883, Schultze[23] writes of two cases under the title of "Ascending Atrophic Paralysis with Fatal Outcome," one in a man of 44, and the other in a man of 30. Immermann[24] in 1885 reported an important case with autopsy. The patient, a man of 22, had an ascending flaccid paralysis of the legs, followed by involvement of an arm and later bulbar symptoms. The autopsy showed changes in the ventral horns. Immermann assumes from this case that a form of anterior poliomyelitis exists which corresponds to the clinical picture of Landry's paralysis.* Various

*"Die Landry'sche Paralyse und die acuta Poliomyelitis anterior nur die graduell verschiedenen Bilder *eines* Krankheitsprocesses darstellen." *Loc. cit.*, p. 305.

other writers on the subject note the fact that in many cases the onset of the paralysis is by no means so stormy as in infants, but they are not thereby induced to exclude them from the general category of poliomyelitis, influenced no doubt by the autopsy findings.

Discussion has also arisen with regard to the differential diagnosis between poliomyelitis of the adult and multiple neuritis. In 1880, Leyden[25], in an exhaustive paper, attempted to elucidate the question by a somewhat elaborate classification of the forms in which poliomyelitis may occur. One form he regarded as essentially due to a peripheral neuritis. Very recently Strümpell and Barthelmes[26] have again reverted to the matter in much clinical detail, calling attention to the fact that the diagnosis of adult poliomyelitis has decreased with the advent of the study of polyneuritis. These writers refer to Müller's[27] monograph on acute atrophic spinal paralysis, in which no case is to be unquestionably regarded as an acute poliomyelitis.

Except in rare instances the differential diagnosis on the clinical side should not present many difficulties provided the term neuritis is used in the sense of an inflammation of the peripheral nerve trunks, and not as another term for neurone degeneration. In the latter case various forms of motor neurone degenerations might easily simulate a poliomyelitis, with or without transient sensory disorder. The following cases are of interest in this connection:

Case V.—W. P., a farmer, nineteen years old, came to the Massachusetts General Hospital in March of this year. About a year and a half before, he had gone to bed feeling well. During the night he had considerable pain in the back, which kept him awake. The following day he had headache, for which he remained in bed. In the afternoon, on attempting to walk, he found that his right leg "gave way" at the knee. For the next two or three days both legs were slightly sensitive to pressure, which persisted in modified form for some weeks. Treatment by electricity and massage did no good, and the right leg gradually decreased in size and became sensitive to cold. Examination a year and half after the onset showed a marked paresis of the right leg. The patient could lift it slightly while sitting, but was wholly unable to extend the leg on the thigh. The sphincters were unaffected. There was no venereal or alcoholic

history; the right knee-jerk was absent; there was no objective disturbance of sensation.

Case VI.—A man of twenty-nine, a temporary patient at the Massachusetts General Hospital, said that ten years before he had had "rheumatism in his knees," extending to his hips, by which he was crippled for six months. The right leg apparently recovered, but the left has remained weak. There was wasting below the knees, and a complete paralysis of the left foot and toes for a year. The general health was good, and at the time of examination there were no sensory disturbances.

Whatever doubt there may be as to the proper interpretation of these cases, in both of which the diagnosis of poliomyelitis was made, it will be generally admitted that cases I and II are typical of what has ordinarily been called poliomyelitis, and certainly could not be brought into the category of polyneuritis.

Temporary disorders of sensation, often of an extreme degree of severity, have frequently been described in the early stages of the disease, no doubt to be explained by the involvement of portions of the dorsal horns in the inflammatory process. This should never lead to an error in the clinical diagnosis, particularly after the acute onset has passed.

Pathological Anatomy.—As our study of the pathological anatomy of the nervous system progresses, it is becoming increasingly clear that we must continually widen the bounds of what we have hitherto regarded as individual diseases. This is particularly true of the group of lesions it is now necessary to consider in connection with a process apparently so clearly defined as poliomyelitis. This disease has, perhaps, occupied a somewhat more stable position than many other affections of supposed definite organic basis, but the time has apparently come for a clearer recognition of its relation to various cerebral and peripheral disorders of so dissimilar a character clinically as peripheral neuritis and acute encephalitis or Landry's symptom-complex and acute ophthalmoplegia. Is poliomyelitis occurring either in the child or adult the same disease as these, and due to the same general causes, or are we dealing with totally dissimilar affections both from the pathological and etiological, as well as from the clinical, point of view? This question may, at present, be answered only in part,

but much light has been thrown upon it by various recent pathological reports.

Regarding poliomyelitis certain facts are now generally accepted:

1. That it is an infection of undetermined character, limited to the region of the ventral horns of the cord.

2. That the pathological alterations found are consistent with the theory of a primary inflammation and a secondary destruction of nerve-cells.

3. That the ultimate changes may closely resemble or be identical with those observed in primary degenerations of nerve elements.

4. That the symptoms resulting tend to be of sudden onset, capable of some degree of improvement, motor in type and unassociated with permanent sensory disturbance.

Even these fundamental statements have by no means been universally accepted, due, no doubt, in great measure to the widespread influence of Charcot. Following the earlier writers on the subject, Heine, Cornil, Duchenne, Prevost, Cuming and others,* Charcot and Joffroy[29], reporting a case of poliomyelitis twenty-five years after its onset, advanced the theory that the ventral nerve-cells are specifically and primarily involved, differing only from progressive muscular atrophy in being attacked more suddenly. With the material at their disposal this supposition was entirely natural. It is, however, somewhat remarkable that certain writers should later have adhered to this view, in spite of much better opportunities for study. Among these is Rissler[29], who had the exceptional opportunity of studying microscopically five cases, three of which were very recent, occurring in the epidemic described by Medin[30]. For reasons which are not altogether obvious, Rissler concludes that in the recent cases, in spite of cellular exudation in combination with degeneration of nerve-cells, the primary affection is of the nerve-cells. In 1893 von Kahlden[31], unconvinced by many apparently conclusive papers maintaining the existence of a primary inflammation, reverts again to primary degeneration of nerve-cells as the essential lesion. He bases

*See Bibliography.

this opinion upon the examination of material from persons in whom the paralysis had been of long standing. In decided opposition to this point of view stands an array of painstaking articles, of which the following are the most important: Roger and Damaschino[32], Schultze[33], Turner[34], Bramwell[35], Drummond[36], Williamson[37], Goldscheider[38], Siemerling[39]. All of these writers and others, reference to whose work will be found in the bibliography, are united in regarding the primary lesion as inflammatory, brought about by an unknown etiological factor, acting in the general distribution of the ventral arteries of the cord. With certain variations in the details of the pathological findings, recent observers are essentially united in this point of view. It is easy to understand that the appearances after the lapse of years might closely simulate those found in processes of quite a different origin; for example, progressive muscular atrophy. The question can only be decided by the examination of relatively recent cases, of which there are now enough at hand to form a final judgment. A matter of much more vital interest, at present, is the determination of the problem whether poliomyelitis represents a disease *sui generis*, or whether it has intimate relationship with others, differing from them only in degree and location, and consequently in symptomatology. A study of these questions is best made in the adult form, since the diseases which poliomyelitis may simulate occur more frequently in adults than in children. Considered from the pathological side, omitting for the present any question of etiology, the relation of poliomyelitis to multiple neuritis, Landry's paralysis, myelitis, encephalitis, poliencephalomyelitis, and progressive muscular atrophy needs to be studied.

Multiple Neuritis:—On the clinical side the possibility of confusion in the diagnosis between poliomyelitis and polyneuritis has long been recognized and discussed. Allusion has already been made to the important papers of Leyden and Strümpell and Barthelmes bearing on this point. However difficult, at times, the clinical diagnosis may be, the histological changes in the two conditions would hardly admit of question if we regard the lesion of poliomyelitis as in all cases a primary inflammation of the ventral horns within a definite arterial area.

If the term neuritis be applied to peripheral neurone degenerations manifesting themselves only in the axones, no confusion could arise in the histological appearances; if the peripheral nerves show evidence of inflammation without destructive lesions in the ventral horns there is likewise no difficulty. If, on the other hand, inflammatory changes of the ventral horns occur with consequent changes in peripheral nerves, the anatomical diagnosis of poliomyelitis is apparent. So far as we are aware no case has been published in which a coincident inflammation of ventral horns and peripheral nerves has occurred, indicating a common origin. In the absence of such a case it is fair to assume that the etiology of the two conditions is essentially different, a point to which we shall revert later. A mere difference in the degree of strength of the irritant is not sufficient to account for the difference in location of the consequent inflammatory reaction. Redlich[40] on the other hand inclines to the view that poliomyelitis and polyneuritis are the same affection with different localizations.

Landry's Paralysis:—As has been suggested, the tendency to find a pathological anatomy for a group of symptoms resembling in some respects poliomyelitis has led to relatively frequent descriptions of the lesions of poliomyelitis under the heading of Landry's paralysis. Judging from the illustrations, as well as the description of the course of the disease and the lesions, Bailey and Ewing's[41] case is of this character. In this case there was sudden loss of power in both legs, followed the next day by paralysis of one arm and paresis of the other. Sensation was unaffected; the reflexes were lost; there was diminished faradic response in the paralyzed muscles. On the clinical side such a history corresponds perfectly to adult poliomyelitis with cerebral involvement and very imperfectly to the symptom-complex described by Landry. The gradual invasion of muscle groups without electrical changes, supposed to be characteristic of Landry's paralysis did not occur. On the pathological side "the lesion consisted in intense congestion of all the blood-vessels especially of the ramifications of the central branch of the anterior spinal artery," and again, "at some levels congestion and exudation were so intense that nothing could be

distinguished but the round-cell infiltration and the detritus of neuroglia and ganglion cells." Various degenerative changes in nerve-cells were also, naturally, found. These are unquestionably the exact lesions of poliomyelitis as described frequently in children by entirely competent observers, and less often in adults. They cannot be brought into the category of Landry's paralysis, unless that vague term be enlarged to include poliomyelitis, which seems manifestly undesirable. Thomas'[42] Case I is likewise a case of acute anterior poliomyelitis in the adult, described in his paper, entitled "Two Cases of Acute Ascending Paralysis with Autopsy." The diagnosis is so made in the description of the case, and a study of the sections, with which Dr. Thomas has kindly provided me, leaves no doubt of the fact that it is not properly classified under Landry's paralysis. A similar case of much interest has been published by Greene, Wilson and Rothrock[43] as "A Case of Landry's Paralysis." There was high temperature, followed by sudden paralysis of legs, quickly extending to arms. Death, after forty-one days of artificial respiration. Microscopic examination showed a violent inflammation in the ventral horns, extending the entire length of the cord, leading the authors to speak of the lesion as a "parenchymatous ascending acute poliomyelitis." In 1875 Webber[44] in a paper on myelitis deprecated the use of the term acute ascending paralysis as misleading. In this connection articles by Schultze[45], Immermann[46], Jagiç[47], Mills and Spiller[48], as well as those quoted above, are of much interest. As expressed in a former paper[49], it seems altogether desirable to abide by pathological findings, unless a better criterion be offered. If this had been done, we should certainly have heard much more of the supposedly rare poliomyelitis of the adult.

Myelitis occurring in its typical form as a more or less complete transverse lesion of the cord of inflammatory character should not often be confused with, or mistaken for, poliomyelitis. That transitional forms of inflammation may occur must be conceded. It has long been recognized that the lesions of poliomyelitis may extend beyond the confines of the ventral horns, but the greatest intensity of the process has usually been fairly

limited to those areas (Roger and Damaschino[50], Turner[51], Eisenlohr[52], Rissler[53], Goldscheider[54]. A very important case in this connection is one described at length by J. J. Putnam[55] in 1883. The patient was a woman of twenty-two, who was suddenly attacked with disorders of sensation, followed by muscular wasting, the right arm and hand being especially affected. A diagnosis of adult poliomyelitis was made. Microscopic examination of the cord showed that both ventral and dorsal horns were involved, the lumbar cord being less affected than other regions. The blood vessels were thickened, deeply congested, and there was extravasation of cells. The inflammation was acutest in the white matter, many of the fibers of which showed degenerative changes. The ventral nerve roots were degenerated to a greater degree than the dorsal. There was a moderate amount of leptomeningitis. Putnam concludes from the pathological findings that the disease might better be designated, "diffuse myelitis." It is a matter of regret that the peripheral nerves were not examined. From our present point of view the case could with difficulty be placed in the category of acute poliomyelitis. It illustrates better than any case of which we have knowledge, the extension of an inflammation, very similar in general character to that which occurs in poliomyelitis, over a considerable area of the cord, and goes far toward proving the existence of transitional forms of disease. The coincident occurrence of a meningitis is also important.

Encephalitis and Allied Conditions.—In many cases in which a poliomyelitis is the predominant feature in the clinical picture, cerebral involvements of more or less severity have been described, and many of them confirmed by autopsy. In Case III of this paper, an unmistakable poliomyelitis was associated for about two months at the onset of the disease with a profound disturbance of consciousness, without involvement of cranial nerves which finally disappeared, leaving no defect. The possibility of a mild encephalitis must certainly here be considered, though we have no means of proving its existence. Involvements of cranial nerve nuclei have been reported many times as poliencephalitis, superior or inferior, or ophthalmoplegia, without involvement of the cord. That the character of these

various processes is similar is shown by the fact that many carefully studied cases of poliomyelitis, especially in the adult, give evidence of a slighter, but perfectly definite inflammatory reaction throughout a part or whole of the brain stem. Such observations have been made by Gombault[56], Rissler[57], Redlich[58], Greene, Wilson and Rothrock[59], Jagiç[60], Bülow-Hansen and Harbitz[61], and on the clinical side notably by Caverly[62], and Medin[63], who studied the two best recognized epidemics of the disease which have hitherto occurred. In these cases the cord affection was, for the most part, predominant, and what changes were found in the oblongata were regarded as subordinate in intensity. Bailey and Ewing's case of Landry's paralysis, to which we have previously alluded, is of this type; in that case lesions were also found in the cerebellum. A still more definite connecting link between the cerebral and spinal affections is to be found in the rare cases of poliencephalomyelitis, a few of which have been carefully reported with subsequent autopsy. Under the heading "A Case of Poliencephalomyelitis in an Adult, Presenting the Clinical Picture of Landry's Paralysis, and Fatal in Thirty-eight Hours After First Definite Motor Symptoms," Sherman and Spiller[64] have recently reported a characteristic instance of combined cord and brain lesions. The patient, a man of twenty-one, was taken with headache, vomiting and slight rise of temperature. Two days later walking became difficult through weakness of the legs; the next day respiratory paralysis set in followed by weakness of the arms, the legs being almost completely paralyzed. The mind remained clear and sensation was unaffected. Death from paralysis of respiration. The pathological examination showed organisms of the colon group from liver and kidneys; engorgement of vessels of the cord in all regions; cellular infiltration of gray matter, meninges and spinal roots; less in white matter and less in dorsal than ventral horns; hemorrhages in spinal gray matter; degeneration and loss of ventral nerve cells; ventral spinal roots normal. The oblongata was similarly affected, but to less degree; degenerattive changes in nucleus ambiguus; vessels of pons, peduncles and cortex distended, but with slight perivascular infiltration. From a study of this and related cases, Spiller

concludes that Landry's paralysis may be due to poliomyelitis; that poliomyelitis may be due to a variety of organisms, and that inflammation occurs in the meninges and dorsal horns as well as in the ventral horns; that meningitis is not uncommon; that poliomyelitis in the adult is the same disease as in the child; that it bears a close relationship to non-purulent encephalitis and to poliencephalitis superior. With these deductions we must essentially agree.

The chief interest of this valuable study of Spiller's is the demonstration that widespread inflammation of the central nervous system may occur, leading even to a meningitis, and giving rise chiefly to motor disorders not confined to the cord. The analogy to uncomplicated poliomyelitis is apparent, and the differences are significant of the wide association poliomyelitis may have with inflammatory disorders of other portions of the nervous system.

The following case is a striking clinical example of this fact:

Case VII.—A man of twenty-seven, formerly able-bodied, after an attack of influenza, was paralyzed in both arms. This paralysis improved somewhat, leaving a high degree of muscular atrophy, without disordered sensation. Associated with this paralysis of the arms was a complete paralysis of the external rectus on the right; a partial paralysis on the left; paralysis of the right motor fifth nerve, with some disorder of sensation in the distribution of the sensory branches; paralysis of all branches of the right seventh; deafness on the right; hypoglossal and vago-glossopharyngeal group also involved to a certain degree. The patient when last seen was improving slightly.

In this case there was clearly an inferior poliencephalitis with a cervical poliomyelitis.

Sub-acute and Chronic Poliomyelitis; Progressive Muscular Atrophy:—That poliomyelitis in the adult may take several days for its complete development has already been stated; a more sudden onset than this is rare. This is, no doubt, to be explained by the greater natural resistance of the adult nervous system to toxic influences than the child's. Even a very acute meningitis or pneumonia takes a certain time for its development, and there is no reason whatever why the same should not be true in the ventral horns of the cord. The virulence of

the infection must influence the rapidity of development of the symptoms. Before any adequate explanation of this may be expected we must have a far more detailed knowledge of etiology than is now at hand. In the meantime on the clinical and pathological side a few cases have been described on such good authority that the types of sub-acute and chronic poliomyelitis have been introduced into the nomenclature. In 1888, Oppenheim[65] described a case of three years' duration in a woman of about fifty, in which death finally resulted in consequence of a gradual increasing paralysis, beginning in one arm. There was loss of ventral nerve-cells, without atrophy of ventral roots or peripheral nerves. The lesions were sharply localized in the ventral horns. Before this in 1882, Eisenlohr[66] expressed himself as confident of the existence of a true sub-acute and chronic poliomyelitis. In 1893 Goldscheider[67] in an exhaustive paper discusses the subject of chronic poliomyelitis, regarding its inflammatory character as doubtful. In general it would appear hardly necessary to separate these cases of gradual onset and fatal termination from the group of progressive muscular atrophy to which they correspond much more closely than to poliomyelitis as ordinarily understood. In the absence of evidences of inflammation this attitude is still further justified, admitting always the possibility of transitional forms which must ultimately be classified on an etiological basis.

From the foregoing discussion of the pathological anatomy of poliomyelitis, it must be evident that the disease, as seen especially in the adult, represents simply a favorite location of an inflammatory process. Sufficient evidence is now at hand to prove that whether cerebral or spinal, the acute inflammations are identical in their histological manifestations, and that we are forced to the conception of poliomyelitis merely as one of a series of more or less sharply localized inflammations.

Etiology:—Speculations regarding the cause of these widespread inflammatory reactions affecting at times every part of the nervous system, are as yet somewhat premature. It is, however, becoming more and more evident that some working basis which goes beyond the findings of the microscope is advisable,

provided it does not conflict with ascertained knowledge. Such an attempt to throw light on the complex relationships of the affections we have been considering, has recently been made by Strümpell and Barthelmes in their paper to which we have alluded: "Ueber Poliomyelitis acuta der Erwachsenen und über das Verhältniss der Poliomyelitis zur Polyneuritis."[68]. After speaking of the difficulty in establishing a sharp distinction between degeneration and inflammation, and discussing the general theories of the toxic causes of pathological processes, these writers offer the following etiological classification of the idiopathic atrophic paralyses:

1. The local acute infectious inflammation of a peripheral nerve—acute local neuritis.

2. The local acute infectious inflammation in the region of the ventral horns of the cord—acute local poliomyelitis of children and adults.

3. The hematogenous—toxic motor nerve-degeneration—the so-called acute polyneuritis in its different forms of origin and distribution.

4. The acute, sub-acute and chronic exogenous (toxic?) degenerations of peripheral motor neurones, including the motor spinal ganglion-cells—usually hitherto known as "poliomyelitis," sub-acute and chronic.

5. The endogenous progressive atrophy of motor neurones —neurotic and spinal progressive muscular atrophy and associated conditions (amyotrophic lateral sclerosis, etc.)

Such a classification, imperfect as it must necessarily be, has the merit of placing what exact knowledge we have on a more definite foundation, laying stress on the association of diseases, apparently dissimilar rather than in exaggerating their superficial clinical differences. The nature of the toxemia, and the reasons for its selective action remain wholly beyond the field of our present knowledge. It is a possible assumption and in a measure, at least, justified by the facts, that the striking differences between degeneration on the one hand (progressive muscular atrophy), and violent inflammations on the other (acute poliomyelitis), lie in the intensity as well as in the character of the exciting toxic cause plus the vulnerability of the affected

individual. According to this grouping likewise, adult poliomyelitis simply occupies one position in a wide series of related conditions.

Conclusions:—The following conclusions as a practical clinical guide and as a working basis for further study seem justified.

That adult poliomyelitis is a well-marked clinical affection, characterized by initial fever, rapid onset of usually extensive paralyses, motor in type, with a tendency toward recovery, though often resulting fatally, from respiratory paralysis. That the disease has frequently been confused with multiple neuritis and so-called Landry's paralysis.

That its anatomical basis is a primary inflammation in the distribution of the ventral arteries of the cord, leading to a destruction of nerve-cells.

That this inflammation is rarely sharply limited to the ventral horns, but extends into the dorsal gray matter, the surrounding white matter and at times into the oblongata.

That there is no sharp line to be drawn between these lesions and still more extensive ones giving rise to totally different clinical pictures *e.g.*, encephalitis, poliencephalitis, poliencephalomyelitis.

That, therefore, anatomically the disease is much less sharply characterized than it is clinically. That its final place must be determined by a study of its cause or causes, as related to various other degenerations and inflammations of the nervous system.

That from the practical point of view it is well to consider those cases poliomyelitis which show a flaccid atrophic paralysis of sudden onset, with definite anatomical changes limited to the ventral horns of the cord and their immediate vicinity.

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[63]*loc. cit.*
[64]*loc. cit.*
[65]Arch. f. Psych., xix, 381, 1888; also Arch. f. Psych., xxiv, 758, 1892.
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SUMMARY OF CASES OF POLIOMYELITIS REPORTED IN THE LITERATURE.

In the following list of titles the important literature of poliomyelitis is given, with special reference to its occurrence in the adult, and to the pathological anatomy both in adults and children, together with certain related collateral papers. No attempt has been made to collect minor clinical reports. Various important papers bearing indirectly on the subject of poliomyelitis, especially in the adult, have recently appeared under the title of Landry's Paralysis, or Acute Ascending Paralysis. These are referred to in the foregoing text.

Duchenne: "Electrisation localizée," 1st ed., 1855.

Heine: "Beobachtungen über Lähmungszustände der unteren Extremitäten." Stuttgart, 1840.

Also: "Ueber spinale Kinderlähmung," Stuttgart, 1860.

Cornil, V.: "Paralysie infantile; cancer des seins; autopsie: altérations de la moelle épinière, des nerfs et des muscles; généralizations du cancer." Comptes Rend. de la Soc. de Biol., 187, 1863.

The first case reported with autopsy; onset in child of 2; death at 49. Loss of nerve cells and atrophy of muscles noted.

Duchenne (de Boulogne) fils: "De la paralysie atrophique graisseuse de l'enfance." Arch. gén. de Méd. II, 28, 1864. Clinical report of cases. Teeth regarded as important in etiology.

Prevost: "Observation de paralysie infantile; lésion des muscles et de la moelle." Comp. rend. de la Soc. Biol., 215, 1865.

Woman of 78: poliomyelitis in infancy. Loss of nerve cells; overgrowth of neuroglia, asymmetry of cord; reduction in size of left ventral horn.

Johnson and Clarke: "On a Remarkable Case of Extreme Muscular Atrophy with Extensive Disease of the Spinal Cord. Medico-chirurg. Trans. Lond., li, 250, 1868.

Man of 32, the disease dating from first year. Good description of pathological changes; artefacts misinterpreted. Opinion advanced that atrophy of cells is the cause of changes in the muscles.

Cuming: "Case of Extensive Paralysis from Morbid Condition of the Spinal Cord, Probably Congestion." Dublin Quart. Jour. of Med. Sciences, xlvii, 471, 1869.

Case without autopsy, in man of 40; possible alcoholic neuritis.

Charcot, Joffroy: "Cas de paralysie infantile spinale avec lésions des cornes anterieures de la substance grise de la moelle épinière." Arch. de Physiol. iii, 134, 1870.

Poliomyelitis at 7, death at 40, atrophic changes in ventral horns; degeneration of ventral nerve roots. The theory advanced that ventral nerve cells are specifically and first involved, as in progressive muscular atrophy, but more suddenly.

Parrot, Joffroy: "Note sur un cas de paralysie infantile." Arch. de Physiol. iii, 309, 1870.

Autopsy, showing loss of nerve cells and atrophy of one ventral horn with vascular infiltration.

Roger, Damaschino: "Récherches anatomo-pathologiques sur la paralysie spinale de l'enfance." Gaz. méd. de Paris, xxvi, 457, 480, 505, 541, 576; 1871.

Roger, Damaschino: Same title, Comp. rend. de la Soc. Biol. 49, 1871.

Elaborate paper, with a review of previous literature. Three cases in children reported with autopsies 2, 6 and 13 months after onset. General conclusions that lesion is primary in the cord, and secondary in muscles and nerves; that the process is an inflammatory softening of the nature of a myelitis.

Förster: Jahrbuch f. Kinderheilk. Neue Folge, V, 118, 1872.

Brief statement of two autopsies in infants; changes in peripheral nerves in one, and in cells and nerves in the other.

Chalvet: Thèse de Paris, 1872.

Gombault: "Note sur un cas de paralysie spinale de l'adulte, suivi d'autopsie." Arch. de Physiol., v, 80, 1873.

First adult case with autopsy reported. Woman of 67, typical sudden onset with fever followed by extensive paralysis of the extremities; death at end of 7 years. Post-mortem showed involvement of ventral horns, some degeneration of ventral roots, and slight involvement of oblongata; lesions not regarded as primarily inflammatory.

Petitfils: "Considérations sur l'atrophie aigue des cellules motrices (Paralysie infantile spinale; paralysie spinale aigue de l'adulte)." Paris, 1873.

Roth: "Anatomischer Befund bei spinaler Kinderlähmung." Virchow's arch., lviii, 263, 1873.

Child of 2, death 11 months after onset. Usual changes in ventral horns; dorsal horns and white matter slightly involved.

Bernhardt, M.: "Ueber eine der spinalen Kinderlähmung ähnliche Affection Erwachsener." Arch. f. Psych. iv, 370, 1874.

Three typical cases in adults; no autopsies. Allusion to the fact that the four extremities are usually affected in the adult.

Frey: "Ueber temporäre Lähmungen Erwachsener, die den temporären Spinalähmungen der Kinder analog sind und von Myelitis der Vorderhörner anszugehen scheinen." Berl. klin. Woch., xi, 3, 13, 28, 1874.

Clinical report of cases of temporary paralysis, with reference to observations of Kussmaul, Deutsch, Arch. f. klin. Med., i, 506, 1866.

Frey: "Ein Fall von subacute Lähmung Erwachsener—wahrscheinlich Poliomyelitis anterior subacuta." Berl. klin. Woch., xi, 549, 566, 1874.

Erb: "Ueber acute Spinallähmung (Poliomyelitis anterior acuta) bei Erwachsenen, und über verwandte spinale Erkrankungen." Arch. f. Psych. v, 758, 1874.

Valuable clinical paper with report of seven cases. Objection raised to the diagnosis "acute ascending paralysis," in many cases probably poliomyelitis.

Webber, S. G.: "A Contribution to the Study of Myelitis." Proc. Am. Neurolog. Ass'n, 55, 1875.

Alludes to involvement of white matter in poliomyelitis. Deprecates use of term, "acute ascending paralysis."

Leyden: "Beiträge zur pathologischen anatomie der atrophischen Lähmung der Kinder und der Erwachsenen." Arch. f. Psych. u. Nervenkrankh., vi, 271, 1875.

Four cases reported with late autopsies; one case without autopsy, and others quoted. Various pathological processes described, the identity of which is not definitely made out.

Cornil, Lépine: "Sur un cas de paralysie générale spinale antérieure subaigue, suivi d'autopsie." Gaz. med. de Paris, No. 11, 127, 1875.

Man of 27, gradual onset, so-called subacute poliomyelitis. Alterations found in cord.

Schultze, F.: "Zur Lehre von der spinalen Kinderlähmung und der analogen Lähmung der Erwachsenen." Virchow's Arch. lxviii, 128, 1876.

Onset at 3; death at 23. Changes in lateral tracts and Clarke's columns, in addition to usual alterations, believed to be the result of an inflammatory process. A second case, with autopsy, resembling in early course a rapid Landry's paralysis.

Dejerine: "Note sur deux cas de paralysie infantile." Le Prog. méd. vi, 423, 1878.

Two cases with autopsy, in children, believed to confirm Charcot's view of the parenchymatous nature of the disease.

Taylor, F.: "Spinal cord from a Case of Infantile Paralysis."

Trans. Patholog. Soc. Lond., xxx, 197, 1879. Description of usual changes.

Turner, F. C.: "A Portion of the Spinal Cord, with Drawings and Microscopical Specimens, from a Case of Acute Anterior Poliomyelitis in a Child, Fatal within Six weeks from the Onset." Trans. Patholog. Soc. Lond., xxx, 202, 1879.

Child of 2½; typical inflammatory changes found; some involvement of dorsal horns.

Humphrey, H.: "Case of Infantile Paralysis." Trans. Patholog. Soc. Lond., xxx, 211, 1879.

Case with autopsy in a child; duration of disease about 2 years.

Müller, F.: "Die acute atrophische Spinallähmung der Erwachsenen." Stuttgart, 1880.

Eisenlohr: "Zur Pathologie und pathologischen Anatomie der spinalen Kinderlähmung." Deutsch. Arch. f. klin. Med., xxvi, 557, 1880.

Two cases, infants; autopsies 6 and 12 months after onset. Typical lesions; slight involvement of dorsal horns and white matter.

Rank: "Zur Lehre von der Poliomyelitis anterior acuta adultorum." Deutsch. Arch. f. klin. Med., xxvii, 129, 1880.

Two cases, without autopsy; elaborate electrical examinations. Summary of knowledge up to that time.

Leyden: "Ueber Poliomyelitis and Neuritis." Ztschft. f. klin. Med. i, 400, 1880.

Important and exhaustive paper. Attempt to differentiate three forms of the disease, one of which is a peripheral neuritis.

Sange: Thèse de Paris, 1881.

Bramwell, B.: "Microscopical Preparations and Drawings Demonstrated at Meeting of Med. Chirurg. Soc. of Edin. of Pathlogy of Infantile Paralysis." Edin. Med. Jour., xxvi, 745, 1881.

Well illustrated paper, showing characteristic changes in a child's cord.

Eisenlohr: Poliomyelitis anterior subacute cervicalis circumscripta beim Erwachsenen." Neurolog. Ctbltt., 1, 409, 1882.

Man of 52; slowly progressing palsy of five years' standing. Post-mortem, cord showed atrophy of ventral horn cells. E. is positive of existence of true subacute and chronic poliomyelitis.

Putnam, J. J. "Examination of the Spinal Cord in a Case of Poliomyelitis of the Adult, of Two months' Standing." Journal of Nervous and Mental Disease, No. 1, 14, 1883.

Important case; probably better classed as a "diffuse myelitis." [See text of this paper.]

Archambault, Damaschino: "Récherches cliniques et anatomo-pathologiques sur un cas de paralysie spinale de l'enfance, avec autopsie au

vingt-sixième jour de la maladie." Rev. mensuelle des mal. de l'enfance. I, 63, 1883.

"Red softening" in ventral horns.

Sahli, H.: "Zur Lehre von der spinalen Localization. Sectionsbefund bei einer alten Kinderlähmung mit eng localizirter Atrophie." Deutsch. Arch. f. klin. Med., xxxiii, 360, 1883.

Schultze: "Ueber aufsteigende atrophische Paralyse mit tödtichem ausgange." Berl. klin. Woch., xx, 593, 1883.

A case of subabcute onset in a syphilitic of 44; death in 6 months; no autopsy. A second case of insidious onset in a man of 30, death with increasing motor weakness in 8 months. No autopsy. Interesting discussion of Landry's Paralysis.

Money: "The Spinal Cord of a Recent and of an Old Case of Infantile Palsy." Trans. Patholog. Soc. Lond., xxxv., 45, 1884.

Two cases, with unusual changes post-mortem. Fanciful theory offered in explanation of the alterations.

Drummond, D.: "On the Nature of the Spinal Lesion in Poliomyelitis Anterior Acuta, or Infantile Paralysis." Brain, viii, 14, 1885.

Child of 5; death 6 or 7 hours after onset. Vascularity of ventral horns, but not sharply confined to them; minute hemorrhages; free leucocytes; nerve cells swollen, granular and ill-defined.

Immermann: "Ueber Poliomyelitis acuta und Landry'sche Paralyse." Neurolog. Ctblt. iv, 304, 1885.

Man of 22: ascending motor paralysis of the clinical type of Landry's Paralysis. Death; inflammation of ventral horns only.

Friedländer, C.: "Ueber Verkalkung der Ganglienzellen." Virchow's Arch., lxxxviii, 84, 1888.

Three cases with autopsy, one in an adult. Finds a process of calcification of nerve cells which is regarded as a constant change in acute poliomyelitis. See also Virchow's Arch., xi., 620 and l, 304.

Rissler, J. "Zur Kenntniss der Veränderungen des Nervensystems bei Poliomyelitis anterior acuta." Nordiskt med. arkiv., xx, No. 22, 1, 1888.

Five cases, with autopsies, 3 early, 2 old, 1 an adult with death on the eighth day. Conclusion that in acute cases there is cellular exudation and degeneration of nerve cells, but, agreeing with Charcot, that the cellular changes are primary. A long, unnecessarily tedious, but very important paper.

Oppenheim: "Ueber die Poliomyelitis anterior chronica." Arch. f. Psych., xix, 318, 1888.

Gradual motor paralysis in a woman of 53, increasing over a period of three years. Loss of ventral nerve cells, without corresponding atrophy of ventral nerve roots and peripheral nerves. Important case in connection with so-called chronic poliomyelitis.

Kawka: Halle Dissertation, 1889.

Joffroy, Achard: "Contribution a l'anatomie pathologique de la paralysie spinale aigue de l'enfance." Arch. de méd. expériment., i, 57, 1889.

Two cases of poliomyelitis in infancy; death in old age. Typical lesions, including changes in peripheral nerves, muscles and bones.

Rosenberg: Heidelberg Dissertation, 1890.

Williamson: "The Early Changes in the Spinal Cord in Acute Anterior Poliomyelitis of the Adult." Med. Chron., Manchester, xii, 454, 1890.

Man of 22; death at end of five weeks. Inflammatory changes in ventral horns. No microorganisms found.

Medin: Ueber eine Epidemic von spinaler Kinderlähmung." Verhandl., x, Med. Cong. Berlin, II, Abt. vi, 37, 1891.

Epidemic in Stockholm.

Also: Hygiea, xlii, 657, 1890, and abs. in Neurolog. Ctbltt., 397, 1891.

Oppenheim: "Zur Pathologie der chronichen atrophischen Spinallähmung." Arch. f. Psych., xxiv, 758, 1892.

Von Kahlden: "Ueber Entzündung und atrophie der Vorderhörner des Rückenmarks." Ziegler's Beiträge, xiii, 113, 1893.

Reversion to Charcot's view of primary change in nerve cells.

Goldscheider: "Ueber Poliomyelitis anterior." Deutsch. med. Woch., xix, 441, 1893.

Goldscheider: "Ueber Poliomyelitis." Ztschrift. f. klin. Med., xxiii, 494, 1893.

Covers ground of former paper more completely. Opposes Charcot's view but regards inflammatory character of chronic poliomyelitis as doubtful. Best review of the whole subject and allied conditions up to this date.

Lippmann: "Poliomyelitis anterior." Deutsch. med. Woch., xix., 823, 1893. Discussion of Goldscheider's first paper, with report of a case in a child, with autopsy.

Dauber: "Zur Lehre von der Poliomyelitis anterior acuta." Deutsch. Ztschft. f. Nervenheilk. iv, 200, 1893.

Case of a child; autopsy, with inflammatory changes in cord.

Redlich: "Beitrag zur pathologischen anatomie der Poliomyelitis anterior acuta infantum." Wien. klin. Woch., vii, 287, 1894.

Infant; death in ten days; extensive inflammatory changes. Suggests that poliomyelitis and polyneuritis are same affection, with different localizations.

Siemerling: "Zur pathologischen Anatomie der spinalen Kinderlämung." Arch. f. Psych., xxvi, 267, 1894.

Two acute cases in infants; usual inflammatory changes found, with secondary nerve cell alterations.

Caverly: "History of an Epidemic of Acute Nervous Disease of Unusual Type." N. Y. Med. Rec., xlvi, 673, 1894.

A revision of this paper was later published in pamphlet form with the title: "History of an Epidemic of Acute Anterior Poliomyelitis."

In this epidemic the farm animals were also attacked. There were about 125 cases, chiefly in children; the brain was frequently involved. There were no autopsies.

Mund: Erlangen Dissertation, 1897. Same case as reported by Strümpell in 1900.

Grawitz: "Ein Fall von Poliomyelitis anterior subacuta mit Zwerckfelllähmung bei einer Erwachsenen." Berl. klin. Woch., xxxiii, 245, 1896.

Woman of 18, involvement of all extremities, of rather gradual onset. The patient did not die.

Bicknel: "Ein Fall von acuter Poliomyelitis beim Erwachsenen unter dem Bilde der aufsteigenden Paralyse."

Bonn: Dissertation, 1898.

Edward: Thèse de Paris, 1898.

Niedner: "Ein Fall von Poliomyelitis Acuta der Erwachsenen." Münch. med. Woch., xl, 566, 1898.

Previously healthy man of 19; sudden onset; death in three days; no autopsy.

Matthes: "Sectionsbefund bei einer frischen spinalen Kinderlähmung." Deutsch. Zeitschrift f. Nervenheilk, xiii, 331, 1898.

Infant; death in eight days. First description of changes by use of Nissl Method.

Schultze: "Zur Aetiologie der Acuten Poliomyelitis." Münch. Med. Woch. xlv, 1197, 1898.

Child of five;; possible coincident meningitis and poliomyelitis. Weichselbaum diplococcus found in cerebro-spinal fluid; no autopsy.

Jagiç: "Zur Kenntnis der acuten Poliomyelitis der Erwachsenen" (Acute aufsteigende Spinallähmung). Wien. med. Woch., xlix., 394, 1899.

Man of 28, simulation of Landry's Paralysis; inflammatory changes of cord and oblongata.

Bülow-Hansen, Harbitz: "Beitrag zur Lehre der acuten Poliomyelitis." Ziegler's Beiträge, xxv, 517, 1899.

Three children in same family, two deaths with autopsy and usual findings.

Dercum: "Note on a Case of Acute Poliomyelitis in Which the Cerebro-spinal Fluid Obtained by a Quincke Puncture contained a diplococcus, resembling the diplococcus of Sternberg." JOURNAL OF NERVOUS AND MENTAL DIS., xxvii, 116, 1900.

Hoppe: "Poliomyelitis anterior acuta adultorum." Jour. Am. Med. Ass'n, xxxiv., 80, 1900.

Report of two cases observed clinically.

Sherman and Spiller: "A Case of Poliencephalomyelitis in an Adult. Presenting the clinical picture of Landry's Paralysis and fatal in 38 hours after first definite motor symptoms." Phil. Med. Jour., v, 734, 1900.

Man of 21, rapid ascending motor paralysis; death from paralysis of respiration; widespread changes of inflammatory character throughout the central nervous system. One of the most important published cases.

Strümpell, Barthelmes: "Ueber Poliomyelitis acuta der Erwachsenen and über das Verhältniss der Poliomyelitis zur Polyneuritis." Deutsch. Ztschft. f. Nervenheilk., xviii, 304, 1900.

An important paper; for details see text.

EPILEPSY IN ITS RELATION TO CRIME.*

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The older definitions of epilepsy state it to be a "disease characterized by tonic and clonic convulsions and followed by loss of consciousness;" but later conceptions of it show that convulsive movements need not be present, and that consciousness need not be entirely lost.

Without attempting to formulate a definition at once applicable in all cases of so protean a malady, we can construct a better one than the above, and say that epilepsy is a nervous disorder, characterized by some impairment or loss of consciousness, co-incident with some impairment or loss of the power of motor coördination, with or without convulsions, the paroxysms being generally abrupt in appearance and short and variable in duration.

This description recognizes variations and degrees in its two most constant and prominent manifestations; one, the impairment or loss of consciousness, which is either present to some extent or is complete in every epileptic convulsion, and which is by far the more important of the two; the other, the impairment or loss of motor coördination, which may not be present enough to be appreciated, and which need concern us but little at this time.

When we see a convulsion of the grand mal type; hear the agonizing epileptic cry; see the victim fall prostrate to the earth; witness the powerful agitation of the muscular system of the entire body; see the bloody froth about the mouth, the lacerated tongue, the deeply congested face, the upturned eyes,

*Read before the Rochester Academy of Medicine, April 2, 1902.

and later the labored breathing and prolonged coma that nearly always follow; we have seen but one-half the picture, and seeing only that, we are apt to regard epilepsy as a disease that affects the physical to the exclusion of the psychical side—and as a thing of the mind entirely apart—while, as a matter of fact, insanity in all forms scarcely offers a broader field for studying abnormal states of mind than does epilepsy in its sundry and diversified types.

The forms of alienation due to epilepsy, however, are often subtle and inconstant in the extreme, and exceedingly difficult of study and classification.

The effects of epilepsy on the mind are temporary, prolonged, or permanent; temporary including transitory states of psychic disturbance that come with the convulsion, that are inseparable from it because they are essentially a part of it, and that disappear with it; prolonged, including forms of psychic disturbance that precede or follow the convulsion, or that coëxist with it; while permanent forms combine all these and include all kinds of fixed insanity occurring in the epileptic independent of his convulsive periods.

The continuously insane epileptic is not usually liable to commit crime, his lunacy being well enough marked to secure his restraint in an institution for the insane. He has passed out of the less constant state of scientific insanity into that necessary for the disease in the eyes of the law.

Such epileptics mostly have some form or degree of dementia; the factors determining the latter being the duration of the epilepsy; its type, frequency and severity, and the original stamina of the individual.

Severe grand mal attacks, that occur daily, or oftener, that always involve the "organs of the mind," and that begin early in life in a subject already weakened by inherited tendencies, may produce complete epileptic dementia in two or three years; while in other cases in which the attacks occur just as frequently and are just as severe, but in which they do not primarily affect the "organs of the mind," and occur in persons of a better stamina, marked mental enfeeblement may be postponed for years, some cases even going through a long life and having a fair degree of intelligence in the end.

It is difficult to estimate the number of epileptics who become permanently insane in a legal sense, that is, who are continuously unable to distinguish between right and wrong, but I should place it at not less than 15 per cent. to 20 per cent.; and it is worthy of note that the epileptic who becomes permanently insane does not lose his epileptic indentity; he continues to have convulsions, and it is the rule for his insanity to become more marked just before his seizures, his irascibility and tendency to violence increasing sharply at such times, invariably to decline with the passing of these periods.

The condition of dementia is characterized by inaction, mental and physical, nearly all faculties appearing to be suspended or destroyed; and, while it is true that permanently insane epileptics are generally harmless, because they are apt to be under proper restraint, it sometimes happens that they are kept at home too long, those about them being misled by a false sense of security, until the cycle of morbid inaction has run its course and there breaks forth, like a thunder clap from a clear sky, an outburst of great violence, full of danger to the patient and to all about him.

While in this condition they are prone to commit unpremediated overt acts or crimes; and, if such a thing were possible, we ought to hold them to be twice removed from any degree of responsibility, for they made absolutely no use of any of the faculties of the mind in planning the deed and were powerless to stay the act of a hand robbed of its controlling force in the brain.

In this state the epileptic stands forth a curious anomaly under the law of irresponsibility, with consciousness subverted or destroyed, and motor coördination a disorganized force free from any law of control.

The epileptic subject to prolonged periods of mental disturbance, and not continuously insane, would be a less difficult medico-legal problem were his epilepsy only constant in type. But this is not the case, for radical changes in this respect are exceedingly common, and always possible in every case.

The same individual may have a classical grand mal seizure to-day, and a petit mal or psychic attack to-morrow. The con-

dition of irresponsibility that always attends a grand mal seizure is self-evident and distinct. So long as the fit lasts there can be no conscious action on the part of the mind. The main thing to determine is when the fit ceases, and in many cases it does not do so with the passing of the convulsion and subsequent coma, for there frequently follows a subconscious state lasting from a few minutes up to several days, or even weeks, during which the patient is wholly automatic. He becomes a mere machine. He is fully able to go about as usual, to properly care for his person, take his meals, make his bed, dust and sweep his room, and do sundry other commonplace things it was his habit to do before the attack; and yet be all the while absolutely ignorant of what he is about. His mind is a blank, while his bodily actions go uninterruptedly on; and should that man commit a crime while in such a state, he ought no more be held responsible for it than a railway engine would be for killing a person while it was running away without a driver in its cab. A person in such a state could have no criminal intent.

This same curious and interesting phenomenon of automatism may appear independent of attacks that disturb motor coördination, when it is called "psychical epilepsy," or the "psychical epileptic equivalent," meaning by the former a seizure that affects only the mind, causing a blank of variable duration in its operation, and by the latter, a frenzied state of great mental excitement that may last for several days, or subside in an hour or so, and which is not accompanied by convulsions of any sort.

Clinically the psychical epileptic equivalent is much like acute periodic insanity, the only difference being that it occurs in persons subject to epilepsy and is credited with taking the place of a regular epileptic seizure.

It is customary to plead "temporary insanity" in criminal cases in persons who do not have epilepsy, and in which the period of homicidal madness lasted only long enough to cover the commission of the act.

The duration of mental disturbance in psychical epileptic equivalent states is longer, lasting from a few minutes up to several days or weeks. They are never momentary, passing

with the plunge of the knife or the report of the pistol; at least such is my conviction at this time, for in a study of over 1,300 cases I have not seen one of this type that lasted so brief a space of time.

Established proof of the presence of the psychical epileptic equivalent ought to settle the absence of responsibility beyond reasonable doubt.

The greatest medico-legal problem connected with epilepsy is encountered in its purely psychical forms—those uncomplicated by any motor disturbance; that make no rude sign of their approach; that sometimes last for hours, or days, or even weeks, and finally pass away as silent as they came.

A. B. I., a man of 39, a patient at the Colony since 1897, illustrates this type of the disease very clearly. On being asked "Do you sometimes have thoughts come into your mind very suddenly that tell you to do something you know to be wrong?" he answered, "No, sir; I'll tell you what I think answers the same purpose to which you refer. The nearest that I can come to it and the most marked one that I ever noticed was just like this. I was at work in the morning on one end of a row of corn and I got three rows or more set up. I looked at my watch and it was half past seven. All of a sudden I felt a queer sensation, then I said, 'There, you have got to look out or you won't know what you are doing in a minute.' That is the last I knew until I came to myself and I was clear across the corn patch (fully ¼ of a mile away), and had three rows of stalks set up. During all that time I wasn't conscious at all, and I wouldn't have known anything about it only I was over there alone, and the work was done." This particular seizure was witnessed by an attendant, who reported it at the time.

M. S., a woman of forty, sat in my office addressing envelopes from a printed list, and addressed six exactly alike, showing much embarrassment when she came to realize what she had done. I happened to glance at her at the time and there was nothing to indicate the presence of a seizure save a slight pallor of the face and a staring expression of the eyes.

P. DeM., another patient, whose duty for several months it has been to sweep and clean certain rooms at 7:30 A.M. ev-

ery day, will have a seizure in the afternoon, or evening, pull out his watch and say, "Well, its half past seven and I must get to work," suiting his action to the words by getting his broom from the closet and going about his customary task in the usual manner, only to "wake up" a little later and appear chagrined at what he has done.

It is never safe to oppose an epileptic while he is automatic, as he is apt to become combative and dangerous.

L. L., a former patient at the Colony, had a petit mal seizure in an elevated railroad train in New York. By the time the guard reached him he seemed to be all right, but when the time came for him to leave the car some minutes later, he made no move to go. The guard took him by the arm to pull him towards the door, when the patient struck at him violently and a rough and tumble fight followed. An officer was called and locked the patient up. When he finally came to his senses he was greatly mortified at being in jail, and on showing his card of epileptic seizures, was permitted to go. He was unconscious when he struck the guard; had no intent whatever to do wrong, and was entirely blameless.

W. D. M. entered the Colony at the age of thirty-eight, having been an epileptic two years only. He was a man of remarkable talent; a graduate of Hamilton College and an honor man in his class; but who for ten years prior to the beginning of his epilepsy dissipated greatly in every way. He suffered from marked port-convulsive automatism, the condition often lasting several days.

If let alone, he would pass through this period all right, all the while performing with much accuracy his daily work of making beds, sweeping floors, cleaning lavatories and polishing brasses.

Once while in an automatic state, he was accidentally struck by another patient who was having a seizure, and he proceeded to beat the man most severely. On regaining consciousness some hours later, he had no knowledge whatever of the assault and was greatly disturbed over what he had done.

One of the most interesting cases of reputed epileptic automatism that has come under my notice is that of R. F. H., a

man of forty, an epileptic eighteen years, and who entered the Colony early in December, 1901.

His family history is not good, and the patient himself drank and smoked to excess until a year after his epilepsy developed, which was at the age of twenty-one. He is a man of intelligence and was profitably employed as a commercial traveler.

It has been his habit for years to keep a diary, noting therein his epileptic seizures with a certain Greek sign, to conceal the identity of his trouble as far as possible.

He left Rochester September 26, 1901, for a prolonged Western trip, and his diary faithfully records the activities of each day up to December 14th following, when he entered the Colony. The notes in the diary are in detail, giving the names of the places visited; the railroads he traveled over to reach them; the hours of arrival and departure; the names of the firms he called on; the result of these calls; together with frequent notes to show how he felt, how many letters he wrote, and many other things of a personal nature. We will omit these diary notes between September 26 and November 15, and give them from that time on as copied from his diary by the patient himself:

Nov. 15. Little business today. Went to Kalamazoo in afternoon. Wrote two letters. Serious epileptic collapse on the street this morning; at Elkhart.

Nov. 16. Saw Kalamazoo trade. Slight touch epilepsy in evening. Wrote three letters.

Nov. 17. Sunday. Spent the day indoors, writing and figuring. Wrote three letters. Epileptic attack, after a smoke this afternoon.

Nov. 18. Visited trade in Battle Creek, Marshall and Albion. Wrote three letters.

Nov. 19. Did the Jackson and Hillsdale trade today. Not much business. Had attacks of epilepsy both forenoon and afternoon.

Nov. 20. Visited Angola and Auburn. Slept at Columbia City. Wrote one letter. Had epileptic attacks both forenoon and afternoon today.

Nov. 21. Columbia City and Fort Wayne both attended today. Toledo by night. One slight epileptic attack today. Wrote four letters.

Nov. 22. Spent the day in Toledo. Up to Detroit on evening train. Received my long-looked-for "medicine quantity" letter at last. Wrote four letters.

Nov. 23. All day in Detroit. Did little business. Troubled with chills and fever. Took strong dose of quinine at bedtime, also some whiskey.

Nov. 24. Spent all day on correspondence and literature, in hotel. Wrote four letters.

Nov. 25. My diary says: total epileptic collapse.

Nov. 26. My diary says I was at Pontiac, also Detroit, but further than this I know nothing, and have no recollection whatever regarding the day.

Nov. 27. My diary says I got up at 8.30 by my watch, i.e., railroad time. Utterly worn out. No memory left. Saw a doctor. Am to be sent home tonight on M. C. R. R. No. 3 lower birth on sleeper. Night messages sent to my home doctor, my firm and my landlord. (I remember none of this.)

Nov. 28. My diary says: Breakfast on train. It also records $30.00 turned over to my employer. (I have no personal recollection of these items.)

Nov. 29, 30 and Dec. 1. At hospital. (No record or recollection of this.) Dec. 2. My diary says: Up and dressed after dinner. My employer called. I am weak but mentally clear today. No recollection hardly.

Dec. 3. My diary says: Up at 9 a.m. No further record.

Dec. 4. My diary says: Up at 9 a.m. Dressed fully. (From this I infer that I only used my dressing gown on the other two days.) My employer called with the Craig Colony papers, which the doctor signed later.

Dec. 5. Up for breakfast. Back to bed. Up again 10 a.m. Wrote a letter. Had an epileptic attack at 7 P.M. Chewed a thermometer when being put in my mouth to take temperature. (I have a sort of hazy recollection of this—spitting out cut glass, etc., but nothing definite.)

Dec. 6. Up and dressed for breakfast. Paid $1.25 for thermometer. Today's visitors were W. H., Mrs. H., and Dr. J. Wrote one letter.

Dec. 7. Up and dressed 7.30 a.m. Got shaved. Went outside and over home. Saw Mrs. H. Back to hospital for dinner. Van— called 8 p.m.

Dec. 8. Sunday. Three ladies, one man called on me today.

Dec. 9. Depressed. Wrote two letters. No further record in diary.

Dec. 10. Diary says: Epilepsy between 4 and 5 p.m. Chewing severe. Record of four letters written. (Beyond these facts diary does not throw any light, nor does memory recall the day at all.)

Dec. 11, 12 and 13. Back home again. (No recollection of these three days. Diary records two letters received and one written, but memory fails to recall them. And these were vitally important days, too, when I was making arrangements for my sojourn at Craig Colony, and trying, presumably, to straighten out my affairs a little before leaving. I discovered, on my return home for a week in February, that I had given away a number of personal effects to friends, and failure to recall this fact caused a few rather embarrassing blunders. For instance: I calmly arranged to have some furniture taken up to the Colony, which I had previously sold to my landlord.

Dec. 14. My employer accompanies me to Sonyea. Diary makes note of the main facts, but my memory fails to respond when I try to recall them now.

Dec. 23. From the 14th till now—a week—my diary makes note of letters received and answered. Also states that on Tuesday, December 17, I was epileptic all day, and on the 18th there is also the sign of epilepsy, but as far as personal recollection goes, my memory is *nil*. I have a sort of "hazy," "dream-like" recollection of matters during that period, when I try to recall it by going to my correspondence and such like, but am unable to fix things at all definitely. (I have carbon-manifold copies of the letters, which show them to be perfectly normal and sensible, yet my memory fails to retain the incidents referred to in said letters.)

From December 23 till today—over two months—I have been in better health than for years past, and have had but three epileptic attacks. (None of them was serious, and each clearly traceable to exciting or depressing primary causes.)

The main feature noticeable in this somewhat disjointed description of my goings-on from September 26 till today, and which I desire to emphasize, is the practical loss of memory from November 25 till December 23. While my diary keeps a fair record of most of the time, and my mind recalls certain days and incidents dimly, I really seem to know nothing as to my existence during that period, and might as well have been in a comatose condition. Though my letters and comments seem perfectly lucid and rational, I have no recollection of most of the incidents there narrated.

It appears from the foregoing that he suffered a "serious epileptic collapse" on November 15th, and had other isolated attacks daily up to the 20th, when they ceased, only to reappear again in the form of "total epileptic collapse" on the 25th, from which time until December 23d, a period of twenty-eight days, he had a "practical loss of memory."

It does not appear that unconsciousness during these twenty-eight days was absolute and complete; for the patient himself says, with every evidence of honesty and sincerity and without any motive for evasion, that while his mind could recall "certain days and incidents dimly," he really seemed to "know nothing" of his existence during that period.

One of the most remarkable features of the case was the patient's seeming ability to write down certain incidents and impressions acquired while he was in a subconscious state, and yet have no knowledge of the fact later, only knowing that he had done so by seeing his own writing on the pages, or copies of letters he had kept.

Kran (*Psychiatric Wochenschrift,* July 7, 1900), reports the case of a male epileptic, twenty-two years of age, given to wandering impulses, during which he would attempt suicide or try to frighten people by flourishing a revolver in their faces, adding that "all his actions at such times were entirely unconscious, in a legal sense, and the patient had no recollection of their occurrence. The periods of amnesia varied considerably; the shortest duration was four days and the longest twenty-three days. The patient was not mentally sound between the attacks; he was always melancholic. During the wandering impulse he was not responsible for his actions, and therefore escaped punishment."

P. DeM., mentioned above, while in this state will answer a question as to the time of day when he is looking at his watch and do it correctly, but he will have no recollection whatever of it afterwards.

Another patient, J. L., will imitate certain gestures made before him while he is automatic, and yet know nothing of it afterwards. If told to make the gestures without being shown how, he pays no attention to the command.

In the *Journal of Mental Science* for January, 1900, White, of the City of London Asylum, in writing on epilepsy associated with insanity says, "The automatic actions of these patients after fits are remarkable. Whatever their erratic behavior may be, they remember nothing of it when they come to themselves. I am one of those who are of opinion that chronic epileptics should be deemed irresponsible for homicidal acts, having seen many cases of epilepsy marked by brutal violence associated with an absolute mental blank as to all that had occurred."

P. P., a young man of twenty-four, a native of Greece, was indicted and recently tried for murder in the first degree in this city for shooting to death his partner in business. Competent testimony was adduced to show that the defendant and the man he killed had been the best of friends for years; that there was absolutely no motive for the crime; and that the defendant not only came from an epileptic family, but was himself a victim of the disease.

On its face, the murder bore every evidence of a cold-blooded crime. In the presence of witnesses the two men quarreled while trying to adjust an account. Angry words passed between them and ugly insinuations, derogatory to the fiancée of the defendant, were made, when he suddenly drew a revolver and fired five shots, point blank, at his partner as fast as a self-acting revolver would work, three of them taking effect and causing death.

Witnesses testified that the defendant then threw his revolver on the floor and made no effort to escape; and it was also testified that he was heard shortly afterwards to say, "It's too bad; it's too bad!" The shooting occurred about quarter past twelve in the afternoon and the defendant was at once lodged

in jail. Other witnesses testified that after the defendant was in jail and when asked what he was doing there, replied, "I shot George."

In his own testimony on the stand the defendant declared that his mind was a blank from the time of the quarrel in the store until he came to his senses in the jail about two and a quarter hours later, and that he knew absolutely nothing of what transpired in that time.

The murder was committed in April, 1901, and the trial held in February, 1902. In the meantime, two certificates—one from a physician, the other from a priest—were secured from Greece, to the effect that the defendant's father was an epileptic; that he often had "nervous sickness" and would fall down almost daily, and that he had been so afflicted for many years.

I was asked to examine the man and give testimony in the case, and repeated examinations made while he was in jail revealed, among other things, the following:

He was born in Greece and came to America nine or ten years ago. Speaks English quite perfectly; seems to understand readily; answers questions in a simple and straightforward way. Age 24; height 5 feet, 2½ inches; weight 134 pounds. Present occupation, flower merchant. Mother died in childbirth; father living and subject to epilepsy; grand-parents lived to old age, father's mother dying of epilepsy. Has one sister who is living and well. No history of insanity, intemperance, or excesses of any kind in any of his ancestors obtainable, save it is stated that his father becomes mentally disturbed at times as the result of his epilepsy.

Temperature 98.5; pulse 88; respiration 20. Physiognomy: complexion clear and smooth; nutrition good; expression calm and benign; speech clear and distinct, except English not perfect. No anatomical stigmata about the face, cranium, teeth, palate, ears, or limbs. Some anomaly of skin in the way of dense hairy growth over the shoulders, chest and arms. No physiological stigmata in the way of tremors, tics, or nystagmus; nor is there any evidence of mental stigmata of any kind. He does not appear to be erratic in any way, emotional or egotistical. All superficial reflexes, including the plantar, cremas-

teric, abdominal, epigastric and scapular, respond naturally. Of the deep reflexes, that of the left patella is somewhat exaggerated, those of the wrist and elbow being normal. The left pupil responds more slowly to light than the right. There is no ankle-clonus and no impairment of tactile sensation in any part of the body. Vision and taste normal; hearing defective, especially on right side. Measurements of limbs show nothing anomalous. Power of grip coequal, but low in both hands.

I was constantly impressed with the man's evident honesty and sincerity, and his expressions of regret for the crime appeared to be genuine.

He stated that the first epileptic seizure he had that he remembered occurred when he was eight, at which time his father told him of a former attack that occurred when he was six. On one occasion he fell from a single story window while in a fit, cutting his head in four places, three small star-shaped scars now appearing on the foreheard a little to the left of the center, and a fourth one in the edge of the hair above the left temple. He described an attack that occurred in Middletown, Conn., in '95, when he fell downstairs, and others that he had in jail after the murder, the first occurring three weeks after the event. The jailer testified that he saw him three different times in attacks of some sort, and, from what I could learn of them from the jailer, it was my belief that the attacks were epileptic.

The defendant testified on the stand that the last thing he remembered in the store—where the shooting was done—was a feeling as though "a wave of cold air was creeping from his feet upward," then his eyes "got dull and flew about;" he saw "yellow, red and green," after which he remembered nothing. He also declared he had the same sensation on the occasion of the attack in Middletown in '95.

The question arose as to the mental state the patient was in when he fired the shots; and I stated it as my belief that if the patient experienced the initial symptoms of an epileptic convulsion, such as he described as being present at the time, he was in an automatic or subconscious state when he committed the deed and could not, therefore, be held responsible.

The word "aura" signifies "breath," and it is defined as a "sensation like a gentle current of air rising from the limbs or body to the head, a frequent forerunner of an epileptic attack;" while the visual aura, in the form of a play of colors before the eyes, is as common as all the other special sense auras combined. The defendant described the presence of these auras in a manner to credit belief in the truth and sincerity of his statements.

The question also arose as to whether a person in a complete state of epileptic automatism could receive a mental impression that he could retain and carry into the conscious state beyond, and I gave it as my opinion that such a thing would not be possible.

Then came the question, "How could the defendant realize he had done something wrong and seem to regret it (as it was claimed this man did, when he said shortly after the shooting, 'It's too bad; it's too bad'), if he was in an automatic state at the time?" and our contention was that even though the defendant might have dimly realized at the time that he had done wrong, it was no evidence of the possession of his mental faculties, for while in such a state a person may be spoken to and will correctly respond, yet retain no knowledge of it when the subconscious state is gone.

It is, perhaps, no more impossible to speak automatically while in such a state than it is to act so.

After prolonged deliberation, the jury returned a verdict of manslaughter in the second degree, the maximum penalty for which is fifteen years' imprisonment, five and one-half being liable to deduction for good behavior.

If this man was an epileptic and was under the influence of a seizure at the time he committed the deed, it does not seem that he should have been convicted. But in such cases we encounter a somewhat anomalous situation, for while the law recognizes the absence of responsibility in insanity, it fails to do so in epilepsy; and if this man had been found insane, he could have been committed to a proper institution, but being an epileptic, he must either be found guilty and made to suffer for his act, or be acquitted and allowed to go free.

If he was not responsible, it is clear that he ought not to have been convicted; if an epileptic and dangerous, it is equally as clear that he ought not to be at large for he may again do a deed of violence at any time.

Had his defense been insanity, the verdict, in all probability, would have been "guilty, but insane;" and had my connection with the case occurred before the line of defense was established, I would have advised such a course, for it is my firm conviction that epilepsy and insanity are often interchangeable terms and that they may sometimes be so when it is beyond human power to affirm or deny the fact; which, being true, it would seem that, given the knowledge of the existence of epilepsy, the epileptic under trial for crime ought always to have the benefit of a reasonable doubt.

In the *Journal of Mental Science* for July, 1901, Percy Smith, Physician for Mental Disorders at the Charing Cross Hospital, reports the case of a man who murdered his wife and child during the night and who was found at noon the next day sitting on a chair in the middle of the room in which the bodies lay, with his night clothing still on, his eyes closed, his head bent forward, and his hands loosely in front of him. An empty vial which had contained chloroform and oil of cloves was found on the washstand, and there was a smell of the latter drug. There were bloodmarks on the prisoner's clothing and on his arm.

He seemed unconscious, made no reply when spoken to, shouted at or shaken, nor any resistance when he was laid on the floor that he might be dressed to be taken to the police station.

On arrival at the police station at 3:15 P.M., further attempts were made to arouse him, and with the idea that possibly he had taken some poison, a stomach pump was passed, but the fluid that was drawn off merely contained clear gastric juice and did not smell of either chloroform or oil of cloves. At the same time he is reported to have said: "I have been roughly treated; which of my children is dead?" Two hours later he was examined again and appeared to be thoroughly conscious of the position he was in and to be sane, but said he was entire-

ly unconscious of anything that had happened between the time he last applied the chloroform and oil of cloves after going to bed, in order to relieve severe neuralgia, and the time when he became conscious at the police station after the stomach pump had been passed.

He burst into tears and used many expressions of endearment in regard to his wife and child, adding, "We never quarreled in our lives. How could I have done it? I suffer very much from my head, especially at night."

He was fifty years of age, and had entered the army in '70, serving for many years in Egyptian campaigns and was known during that time to suffer from "petit mal de soleil" and "fainting spells." As a soldier his conduct was exemplary and he rose to the highest rank obtainable by a non-commissioned officer.

It was also reported that he had a sunstroke shortly after entering the army, that rendered him unconscious from noon one day until the following morning, and that he became subject to "fits" soon after that.

The physicians called to examine him endeavored to establesh his irresponsibility on account of epilepsy.

In his charge to the jury the judge did not refer to the rules in the McNaughton case, nor mention the question of "right and wrong," "a knowledge of the nature and quality of the acts committed," but stated that the question the jury had to consider, practically the only question, was whether the deed was committed under circumstances which would absolve the prisoner from the full consequences of the crime; adding that when the jury was considering the history of the prisoner they could not shut their eyes to the history of the other members of the family.

The jury returned a verdict of "guilty, but insane," and the prisoner was ordered to be detained during the pleasure of the Government.

Aside from the epileptic's inability to distinguish between right and wrong when under the influence of a seizure, we ought to remember that inhibition in most cases is also impaired or destroyed; a fact that seems to escape consideration in establishing responsibility in epilepsy in this country.

It was recognized in 1887, or '88, by Judge J. M. Somerville, of the Supreme Court of Alabama, in a case in which he gave a decision that repudiated in effect the test for insanity stated in the celebrated McNaughton case in England (which was the ability to distinguish between the right and wrong of the act in question), and held that the true test of responsibility in cases of insanity is the power to refrain from doing the act; in other words, whether the alleged evil doer acted under duress of disease which may have impaired or destroyed his powers of inhibition.

The application of a similar test in establishing responsibility in epilepsy would seem especially fitting and just.

Periscope.

Centralblatt f. Nervenheilkunde.

(1902. February 15, No. 2.)

1. On the History of Katatonia. Erich Arndt.

1. *History of Katatonia.*—Arndt provides us with an excellent history of the psychiatry of the first half of the nineteenth century and of that part of later psychiatry in which have arisen katatonia, and its congeners, hebephrenia, primary paranoia, melancholia attonita, and, more remotely, acute confusion. In Pinel's division of the course of mania into four stages, and in Esquirol's separation of "mania without delirium" from general mania to form part of his "monomania," is seen the impulse which led Kahlbaum to his discovery; for those earlier French observers began to comprehend the distinction between a mere mental-state (excitement or depression) and a true disease-unity. Zeller applied Pinel's course-schema too generally, dividing all forms of insanity into four stages; but his pupil, Griesinger, pointed out a broad distinction between those forms which are curable and those which lead to permanent mental weakness, thus bringing in the question of prognosis which Kahlbaum and, later, Kraepelin still more, made the *raison d'être* of katatonia. The question whether catalepsy and other motor signs are merely complications of, or curious phenomena in, melancholia, or are inextricably bound up with it in a disease-entity, has its analogue in the long discussion of the various paralytic manifestations which before the days of Boyle were regarded as mere "complications" of insanity instead of symptoms of the disease-entity paresis. For just as the tremulous speech and progressive weakness of paresis were minutely described before Boyle's discovery, so various phases of katatonia had been described by several observers before Kahlbaum's classification appeared in 1863. Guislain, in his "Extase," first considered the somatic element, the general motor fixation, which later became so prominent in the concept of katatonia, and the nature of which has been such a bone of contention in relation to the disease. Esquirol, Dagonet and others described "verbigeration;" and Arndt, in a paper on "Chorea and Psychoses" (1868) had dwelt upon catalepsy and echolalia and upon the choreiform movements which, as we now know, characterize katatonia.

This was a period of analysis and gave rise to a number of minor and overlapping subtypes. The reaction began with Fink, in 1876, when he pointed out that many hebephenics betray motor symptoms like those of katatonia, cases which he called "katatonic-hebephrenics." Next Schüle maintained that the *paranoia originaria* of San, is virtually a subtype of hebephrenia, and Kahlbaum (1884) showed the affiliations of hebephrenia with the hereditary insanity or insanity of the degenerates of the French. Then Pick proposed to embrace all "the hereditaries stranded on the rock of puberty" (Schüle's picturesque phrase) under the generic term *dementia praecox*, the Latinized form of Morel's *démence précoce.*

Dasaszkiewicz extended the age-limit of the group to thirty years in order to include the katatonics, and lastly Kraepelin added dementia paranoides, the conception of which may be regarded as a development from the primary paranoia of Westphal, who had from the first (1876)

emphasized the relationships of the latter form to katatonia. It is the merit of Kraepelin to have cemented the group of dementia præcox, not on a basis of delusions nor of motor symptoms, nor of clinical course, but on a basis of prognosis,—the outcome in mental weakness.

Arndt discusses various subsidiary theories. Cramer's as to muscle-hallucinations, Freusberg's concerning the cerebral mechanism of katatonia, etc. Altogether in this "History of Katatonia," Arndt has performed a great and needed service to all who are interested in modern psychiatry. PICKETT (Philadelphia).

Centralblatt für Nervenheilkunde und Psychiatrie.

(1902. May.)

1. The Disposition of Insane Criminals. GUSTAVE ASCHAFFENBURG.
2. Podagra and Neuroses. P. J. KOWALEWSKY.

1. *The Criminal Insane.*—No part of the care of the insane, says Aschaffenburg, presents so many difficulties as that which relates to the disposition of insane criminals. In the asylum now being built in Baden, a pavilion has been set apart for 20 to 25 men of this class. The first observation-section of the kind was established in Prussia; and other asylums followed in rapid succession at Breslau, Köln, Münster, Halle and Graudenz. None of these accommodated more than from 40 to 50 patients. Special asylums for the criminal insane have existed in other countries for a long time, Italy having three of them. It is an embarrassing question whether in such an institution cases shall be placed in which insanity has come on after a long criminal career, or only such as have come in conflict with the laws in the course of an acute attack of insanity. For the latter class the brand of "criminal insanity" seems unnecessarily odious, especially when placed upon the hitherto harmless paretic.

Among 27 insane patients who had committed grave crimes, only 4 had been criminals before the onset of their insanity; and anyhow, the problems connected with the management of the criminal in an asylum rest upon the nature of the insanity, and not so much upon the individual's criminal record. Aschaffenburg thinks that it is best to keep the criminal insane in the general asylums, where, in connection with the universities, a study of criminal psychology promises most to medicine and to law.

2. *Podagra and Neuroses.*—After giving the views of various authors on the subject of the relationship of the uric-acid diathesis to migraine and to epilepsy, Kowalewsky cites several cases, in one of which migraine and epilepsy seemed to replace one another as equivalents. In each of these two neuroses the uric-acid diathesis serves as an exciting cause, though in neither can podagra alone account for the attacks; there is always a basis of hereditary predisposition in the victim of epilepsy or migraine. In senile epilepsy sclerosis of vessels and of nerve-tissue is the cause of the attacks, it is true, but podagra underlies these organic changes. PICKETT (Philadelphia).

Rivista Sperimentale di Freniatria.

(1902. Vol. 28, Fascicle 1.)

This entire volume is given up to the proceedings of the Eleventh Congress of the Italian Society of Alienists, which took place during September, 1901. Abstracts of the proceedings are given. Many of the original articles have appeared elsewhere, and of these a number have been abstracted in the JOURNAL OF NERVOUS AND MENTAL DISEASE.

Rivista di Patologia Nervosa e Mentale.
(1902. Vol. vii, fasc. 4, April.)

1. Study of the Function of the Cerebellum. G. Pagano.
2. Yellow Fever as Etiological Factor in a Case of Progressive Paralysis. E. Belmondo.
3. Chronic Cocainism; Psychic Disturbances in a Family Addicted to Cocainism. A. Soutzo (Junior).

1. *Function of the Cerebellum.*—A description of experiments, covering a year's time, in excitation of the cerebellum of dogs by injection of curare into its substance. The author does not yet feel justified in drawing any positive conclusions as to the functional topography of the cerebellum, yet gives numerous findings to date, showing a universal participation of the cerebellum in psychic, motor, and visceral functions. Within the limits of an abstract, detailed description of manifestations elicited by excitation of different areas of the cerebellum is not feasible, but as illustrative of the phenomena from which the author formulates his theory as to the cerebellar function, may be mentioned muscular contractions, varying in degree from contraction of single groups of muscles to epileptic convulsions, induced by injection of curare into the lateral lobe of the cerebellum. These movements are believed to be accomplished through the medium of the cerebral cortex, as excitation of the above-named cerebellar area failed to elicit motor phenomena after extirpation of the motor zone of the cerebrum. The author holds it unreasonable to suppose that the cerebellum is the seat of the manifold functions which are stimulated through its excitation, the more so, that extirpation of that organ does destroy any of the functions; but, what is of fundamental importance, is the finding of physiologists and clinicians that animals deprived of the cerebellum exhibit premature exhaustion. The deduction drawn is that the cerebellum is the sthenic organ of the nervous system, and in it is specialized to the highest degree that vital property of nerve-cells in general; *i.e.*, the storing up of energy to the various nerve-centers when functionating is brought about through reflex action.

2. *Yellow Fever in Etiology of Progressive Paralysis.*—The description of this case constitutes a fresh contribution to the toxico-infectious, or auto-toxic theory of progressive paralysis. The author maintains that whatever may be the pathogenesis of dementia paralytica, hepatic insufficiency is an important predisposing cause as in other psychopathies, for example, delirium tremens. The causal relation between dementia paralytica and yellow fever in this case seems probable from the following facts: (1) Immediate connection of time between attacks of yellow fever and beginning of paralytic symptoms, there having been no previous indications of paralysis; (2) absence of other cause, particularly almost certain absence of syphilitic taint; (3) severity of the attack of yellow fever; (4) occurrence of various paralyses and muscular atrophies of neurotic origin; (5) fatty degeneration of the liver caused by icteroid infection favoring endogenous and exogenous intoxication, and hepatogenous changes in the mental and nervous functions.

3. *Chronic Cocainism.*—Histories of a family of four, in whom the cocaine habit had been acquired from one member.

R. L. Fielding (New York).

Archiv. für Psychiatrie und Nervenkrankheiten.
(1902. Vol. 35, Part ii.)

1. Old and Recent Brain Researches. Edward Hitzig.

2. A Contribution to the Psycho-pathology of Neurasthenia. A. Pick.
3. A Case of Softening in the Dorsal Portion of the Pons Varolii. Albert Ransohoff.
4. Secondary Degeneration and the Patellar Reflexes in a High Transverse Lesion of the Cord. Edward Winter.
5. A Contribution to the Induced Psychoses. O. Kölpin.
6. A Case of Myasthenia Gravis Pseudo-paralytica. Siegmund Auerbach.
7. (I) A Glioma of the Fourth Ventricle. (II) Degeneration of the Posterior and Anterior Roots in Wasting Diseases, and Conditions of Increased Intracranial Pressure. Ph. F. Becker.
8. Post-mortem Findings in a Case of Hystero-epilepsy. Paul Steffens.
9. Statistical Contribution to the Etiology and Symptomatology of Dementia Paralytica. Raecke.

1. *Old and Recent Brain Researches.*—An exhaustive historical, critical and experimental study of the methods of examination, and the theories relating to the investigation of the brain.

2. *A Contribution to the Psycho-pathology of Neurasthenia.*—Prof. Pick directs attention to a peculiar mental state, rarely observed in neurasthenia, which had been clearly pictured by Morel, who described a case. It is characterized by an excessive sensitiveness, and morbid impressionability. A slight and wholly inadequate cause is sufficient to call forth a violent emotional storm, either of depression or exaltation. This lack of emotional equilibrium is often hereditary, and manifests itself in early life. Physiological examples of this type are represented by the so-called "*Gefuhls-menschen.*" Rarely it may so far overstep the normal boundary line as to come within the category of the psychoses. It is to be distinguished from melancholia and the mental state termed "Luxury of Pity;" as well as the "Anxiety Neurosis" of Freud.

3. *A Case of Softening in the Dorsal Portion of the Pons Varolii.*—A woman, aged fifty-five years, five weeks after an apoplectiform insult, presented the following clinical picture: Paralysis of the associated movements of both eyes in a lateral direction. The left eye-ball was rolled inwards, due to spasm of the internal rectus. Convergence was lost. Paralysis of the left facial with implication of the upper branch. Paresis of the right arm and leg. Paresthesia of the right side of the body, with hypesthesia and hypalgesia. The paresis of the right arm and leg increased and was accompanied by weakness on the left side as well. The right abducens nerve recovered. Death two years later.

Microscopical examination—In the dorsal portion of the pons a cyst was found. The left abducens nucleus, left facial nerve, and left fasciculus longitudinalis posterior, were destroyed. Also the greater portion of the median fillet. The pyramidal tract on the left side was also slightly degenerated. A very slight defect was noted in the right posterior longitudinal fasciculus. Ascending and descending degenerations could be traced in the median fillet; also for a short distance in the posterior longitudinal fasciculus. In the summing up of the case, the temporary paralysis of the right abducens is attributed to a distant effect of the lesion [Fernwirkung]. The contracture of the left internal rectus to irritation of the right posterior longitudinal fasciculus. The slight disturbance of the sensation is explained by the incomplete lesion of the median fillet, or according to some the bilateral representation in the hemispheres.

Zwangslachen, which was a very constant symptom, has been frequently observed in pons lesions. Von Bechterew has emphasized this fact and attributes it to a lesion of the thalamo-spinal tract.

4. *Secondary Degeneration and the Patellar Reflexes in a High Transverse Lesion of the Cord.*—A young man sustained a fracture at the level of the third dorsal about 4 mo. There were the clinical evidences of a transverse section of the cord. At first no reflexes below the lesion were elicitable [shock]. Later, however, the knee-jerks could be obtained by reinforcement, but only with difficulty. The plantar reflexes also returned. During the five days preceding death, no patellar phenomenon could be elicited. Microscopically at the seat of greatest compression about 100 nerve fibers were demonstrable. This return and extension of the patellar reflex is explained by the Bastian-Jackson theory: that the cerebrum and cerebellum are antagonistic, the latter furnishing the muscle-tonus and the cerebrum the inhibitory influence through the pyramidal tracts.

The increase of muscle tonus produced by the Jendrassik was sufficient to allow a slight patellar response. When, however, the destruction of nerve-fibers was so far advanced as to cut off communication with the cerebellum, reflex action was irrevocably lost.

Unusual were oculo-pupillary symptoms on the left side, produced by hematomyelia in the posterior horn and adjacent column of Burdach at the level of the 1 D and viii C segments, and implicating the ciliospinal center.

The following secondary degenerations were of interest:

Below the lesion, Schultze's comma tract could be traced to the 12 D, where the last fibers were in close relation to the columns of Clarke. At the level of the 8 D, and continuous with the comma tract at its caudal portion, a less compact area of degeneration appears, which in lower segments approaches the posterior median fissure, and in the lumbar region assumes the area occupied by Flechsig's oval field; this was traced to the sacral cord along the posterior portion of the posterior median fissure.

This the author considers to be anatomically distinct from the comma tract, and in no wise its continuation. The antero-lateral ascending tract of Gowers was traced by the Marchi method through the medulla and pons, situated first in the angle formed by the facial root and the corpus trapezoides, then passing in the lateral fillet and curving backward around the brachium conjunctivum to the anterior medullary velum, and thence to the worm of the cerebellum. In some of the lower animals this course had been demonstrated by Tooth, Mott and Löwenthal.

5. *Contribution to the Induced Psychoses.*—In Schönfeldt's conception of this form of mental disease the following requirements must be fulfilled. The form of the psychosis and the nature of the delusion must be essentially the same in the primarily diseased and the secondarily injected. That the induced malady is the direct result of psychical injection by the first.

Joerger's statement that the secondarily affected not only accepts fully the delusion implanted, but also contributes to its expansion, still further narrows the clinical picture.

Care must be taken to eliminate cases of simultaneous insanity [Folie Simultanée], and Folie Imposée. The latter form may resemble very closely, and indeed is often an earlier stage of, the Folie Communiqué.

The author reports two cases: (I) Man and wife. Both with psychopathic predisposition. The woman excitable, domineering and dog-

matic. The man sensitive, retiring, susceptible. The type of the psychosis was the *Querulenten Wahnsinn.* The man was the victim of mental dejection, but later was the more active and violent in the toils of the delusion. (II) Two sisters, one blind. When they first came under observation both presented symptoms of paranoia. Identical monomania in both. Could not decide which was first affected. Here the psychopathic predisposition could not be demonstrated. The other important factor, community of interests and intimate social and domestic relations, was present in both cases.

6. *Case of Myasthenia Gravis Pseudo-paralytica.*—A woman twenty years of age, immediately following a severe shock, was seized with great weakness of the limbs, and a feeling of respiratory oppression. The next day appeared double vision, which was soon followed by partial ptosis, weakness of the lips, tongue, face and the muscles of mastication and deglutition. The above group of symptoms were aggravated during attacks of influenza, the menstrual periods, and by any unusual bodily or mental strain. Contrary to the usual course, she was more comfortable during the evening than the earlier part of the day. The ocular excursions, although slower than normal, were not paretic, and phonation was perfect. The fatigue-phenomenon of myasthenia was present in the muscles of tongue, face, eyelids, pharynx and jaws, the repetition of the movement five or six times usually sufficing. In the arms and legs, however, while weak, this phenomenon was wanting, while the myasthenic, electrical reactions were typically present. [A similar observation by Murri.] Slight tendency to incontinence of urine and feces. Patellar reflexes were exaggerated.

As the patient is now thirty-seven years old, this curious affection has lasted seventeen years. Oppenheim, in his monograph, cites a case of fifteen years' duration, as the longest on record.

Following Van Horn's theory, based on frog experiments, that fatigue is due to the accumulation of CO_2, and exhaustion to want of oxygen, inhalations of the latter were attempted, but had to be abandoned owing to the dyspnea.

In conclusion, attention is drawn to the faint resemblance of myasthenia gravis and the severe forms of neurasthenia, especially the spinal myasthenia of Löwenthal; also that diplopia and weakness of articulation is not so very rare in these forms.

7. (a) *A Glioma of the Fourth Ventricle.*

7. (b) *Degeneration of the Posterior and Anterior Roots in Wasting Diseases, and Conditions of Increased Intracranial Pressure.*

I. The patient was a woman aged thirty-eight years. The initial symptoms were vertigo and vomiting. A palsy of the right facial soon became apparent, which increased; partial reactions of degeneration. Then failing vision in the left eye, with atrophy. Choked disc absent during the entire course. The right abducens became paretic, then the left third. To this were added paresthesia of the mouth, difficulty in deglutition, mastication and articulation, sense of taste impaired, olfactory sense was obliterated. Hearing was diminished, especially on the right side, but occurred very late. The left facial became affected. Weakness and heaviness on the right side, but never marked. A few days before death, diminished sensation on the left side.

The irregular succession of involvement of the cranial nerves, Rt. 7, Lt. 2, Lt. 3, Rt. 6, Rt. 5, 9, 12, 11, 8, suggested a diffuse basillar process, rather than a single central growth. This tumor, a glioma, was exquisitely infiltrating in its growth, with no sharp boundaries. It occu-

pied the pons and the upper portion of the bulb, projecting into the fourth ventricle, and producing a moderate hydrocephalus.

Headache did not occur until late. The whole course of the disease lasted about eight months.

The left third nerve was not directly involved by the growth, so that its paralysis is explained by distance-effect (Fernwirkung), or possibly by pressure against the clivus of the sphenoid bone. The cause for general muscular weakness, more especially on the right side, was found in degenerations in the pyramidal tracts and in the cells of the anterior horns and anterior roots. The intramedullary portions, posterior of the roots, were also degenerated.

(b) The author has investigated intramedullary degenerations in various conditions. The Marchi method was used. The series of 17 cases included 3 of brain tumor, hydrocephalus 3, cerebral hemorrhage 4, arterio-sclerosis universalis (uremia) 1, Basedow's disease 2 (1 case was negative), carcinoma 1, pernicious anemia 1, phthisis 1, bone tuberculosis 1.

The degenerations were most conspicuous in the lumbar region, next in the cervical, and least in the dorsal. In the posterior roots and columns the most severe and constant degenerations were present. High grade degeneration in the cells of the anterior horns and anterior roots occur, but less frequently. Rarely these degenerations of centrifugal fibers are present, while absent in the centripetal fibers.

Two theories are advanced for the explanation of these degenerative changes: (a) The mechanical theory of C. Mayer, which is based on the supposition that the posterior roots are subjected to pressure as they penetrate the minute pial constrictions on entering the cord. Increase of the intracranial and intravertebral pressure. (b) The toxic theory of Dinkler.

Hoche favors the mechanical theory, and emphasizes the occurrence of optic neuritis in connection with posterior root intramedullary degenerations.

The Schmidt-Manz theory of optic neuritis has a similar mechanical basis.

On the other hand, similar degenerations, which are not to be distinguished histologically, occur in cachexia and other states unassociated with increased pressure; and the pressure would not explain the degeneration of centrifugal fibers, which occur not infrequently. Again, optic neuritis may be absent where intracranial pressure is undisputed. Here the writer concludes that the pressure-theory alone is insufficient to explain these changes.

8. *Post-mortem Findings in a Case of Hystero-epilepsy.*—For the previous clinical description of the case see Vol. 33, Part 3, of this Archiv.

The patient, a woman, was trephined over the left Rolandic area, in 1898, for epileptiform convulsions. The attacks ceased for a while after the operation, then returned and occurred at monthly intervals. Duration fifteen minutes, unconsciousness uncertain, no amnesia, no cry, general convulsions. There was permanent concentric contraction of visual fields to form and color, total analgesia. On January 10, 1901, sudden development of the status epilepticus [a series of 103 convulsions], was operated on the morning of the 11th. Attacks ceased until evening, status epilepticus again developed, death the following day in coma. The status epilepticus had been produced by a hemorrhage at the base of the brain [rupture of an aneurism?] at the site of the first trephine operation. An old superficial area of softening occupied the

mid-Rolandic region. The general convulsions were attributed to this focus. Jacksonian, motor, or sensory attacks, had never occurred.

9. *Statistical Contribution to the Etiology and Symptomatology of Dementia Paralytica.*—A carefully tabulated report of 110 cases of general paralysis of the insane, observed in Siemerling's clinic in Tübingen, from 1894 to 1901. Of these 92 were men, 18 women. During this time there was noted an annual increase in the number of paralytics, especially women.

Etiology—In 57 per cent. antecedent syphilis was proven; in 23 per cent. probable; the remainder negative or unknown; alcoholism in 25 per cent.; a neuropathic predisposition in 31 per cent.; trauma in six cases; the larger number of cases occurred between the ages of 30 and 50.

Symptoms—The pupillary light reflexes were lost in 58 per cent.; altered in 92.7 per cent., and normal in 7.3 per cent.; inequality of the pupils in 83 per cent.; irregular outline in 69 per cent.; jumping pupils in 4 cases. Paradoxical reactions were noted once. The pupils were either normally contracted or moderately dilated; in six cases widely dilated. In these latter the reaction on convergence was also lost.

In Siemerling's comprehensive statistical study at the Berlin Charité, the light reactions were normal in 32 per cent. These cases were under observation only a short time.

The knee-jerks were exaggerated in 55 per cent.; diminished or absent in 33.6 per cent.; unequal in 18.2 per cent.; normal in 10.9 per cent. The exaggeration or diminution of the Achilles-reflex corresponded in a general way to the knee-jerks. Optic atrophy was present in 15.5 per cent. Romberg's symptom in 19 cases. Tremor of hands in 41 cases. Tremor of tongue in 95 cases. Inequality of facial innervation in 59 cases. Associated movements of the face in speaking, 42 cases. Central deafness 10 cases. Tactile sense disturbed in 16 cases. Pain sense disturbed in 63 cases. Muscular atrophies occurred in 10 cases [no R. D.]. There were apoplectiform and epileptiform attacks in 34.5 per cent., occurring more frequently in cases with exaggerated than with abolished knee-jerks. In 12.77 per cent. there was simple dementia without hallucinations or delusions. Hypochondriacal or exalted ideas were present in 67.3 per cent. Violent and prolonged states of mental excitement in 25.5 per cent. The average duration was 2 years and 4 months. In 2 per cent. of the cases, with evidences of pyramidal tract involvement, the first symptom noted was an unconquerable somnolence. J. R. HUNT, (New York).

Revue Neurologique.

(1902. Vol. 16, No. 6, March 31.)

1. Three Cases of Tabes showing Granular Bodies in the Posterior Regions of the Medulla. PIERRE MARIE and BISCHOFFSWERDER.
2. Vitiligo and the Sign of Argyll-Robertson of Syphilitic Origin. M. A. SOUQUES.

1. *Granular Bodies in Tabes.*—For five or six years the authors have made it a rule in all autopsies of tabes to examine the medulla by means of chromo-osmic staining, either according to the freezing method or that of Marchi. Three cases are described to show the disposition of the granular bodies. The histological examination is given very fully and technically, also the methods of preparation and staining. The authors conclude with the following considerations. It is known that the granular bodies are unmistakably indicative of recent degenera-

tion in nerve sheaths; in cases of too recent lesion for the appearance of this degeneration, they are not found, not until all the degenerated nervous matter shall have been eliminated. From the point of view of the age of a lesion and its degree of evolution, the presence or absence of these granular bodies is very significant. In numerous cases of tabes where granular bodies have been wanting, it may be inferred that the destructive pathological process in the posterior cords have ceased; and clinical observation confirms this. In certain old cases of tabes, in fact, the medullary affection seems to have stopped, and new symptoms are rarely noticed. The authors are justified in considering such as having arrived at a stage of the disease from which no farther progress will be made. Moreover, tabes is far from being the utterly fatal disease formerly described; on the other hand, it seems often to have a tendency to stop spontaneously, sometimes in the first period, sometimes after a lapse of years; and it is this factor which renders difficult the appreciation of therapeutic results.

Although results of clinical observation agree with anatomo-pathological investigation, one point must have reservations: in cases of tabetics who die with no granular bodies in the medulla, there are, nevertheless, some of which appear intermittently with the spasm of pain; however, it cannot be said that this symptom is related to the destruction of the medullary nerve fibers; in the three cases reported, the tabes was progressive and the granular bodies existed. One patient died thirty-four months after the appearance of pain, death being hastened by eschars; the second died five days after pain began, committing suicide by throwing himself from a window; the third died eight years after pain began, two years after the appearance of incoördination, committing suicide by cutting the genital organs. Thus in all, death came so soon that the tabetic progress was not completed, and the facts given were, therefore, established.

It is recommended that all who may have occasion to make autopsies of tabetic patients, especially of recent affection, should be careful to preserve and examine, by chromo-osmic reaction, some sections of the medulla for granular bodies; thus, in time, control may be gained of the facts which shall be learned.

2. *Vitiligo and the Sign of Argyll-Robertson.*—A man of sixty-nine years presented the three following symptoms: (1) Paresis of the left vocal cord with bitonal voice; (2) sign of Argyll-Robertson with bilateral myosis; (3) dischromia. The last, present for about fifteen years, was characterized by hypochromic spots surrounded by hyperchromic areas; it was on the trunk and encroached upon the thighs and buttocks; the upper margin, almost perpendicular to the axis of the body, began about two inches below the breasts; the lower margin extended from the front of the gluteal groove over the antero-internal surface of the thighs. The skin of the genital organs was heavily pigmented and presented near the raphe some hypochromic spots. The skin near the scrotum was thick and edematous, so that the region appeared deformed. When the patient entered he had two long serpiginous papulo-squamous syphilides on the abdomen, extending parallel from one antero-superior iliac spine to the other. These typical syphilides disappeared rapidly and completely after a few hypodermic injections of cyanate of mercury. There were no other significant symptoms. Lumbar puncture remained ineffective, though inducing headache and vomiting.

This man could hardly be considered tabetic, but rather, theoretically, a candidate for tabes or general paralysis, as the sign of Argyll is a very frequent symptom of this. But after years of isolation, no further sign appeared. Although the sign of Argyll is associated with syph-

ilitic affections of the nervous system, hemiplegias, myelitis, etc., still it is the single morbid symptom in some cases. In the present case the sign of Argyll and dischromia were independent symptoms, but related through the common cause, syphilis, affecting the nerve centers. Similarly, in vitiligo of tabetics, instead of trophic trouble of tabes, might it not be interpreted as cutaneous trouble dependent upon syphilis? With this hypothesis tabes and vitiligo would constitute an association of coëxistent affections in the same case, with common etiology, syphilis; from the fact that lumbar puncture gave no result, it is probable that the cephalorachidian liquid had spread through the perirachidian tissues. JELLIFFE.

Revue Neurologique.

(1902. Vol. 10, No. 7, April 15.)

1. Muscular Psychoses. A. JOFFROY.

1. *Muscular Psychoses.*—Psychical functions have been compared in normal and pathological states, and it is not difficult to show the harmony between the different functions of the brain, psychical, motor, sensitive, trophic, all uniting in a way to form one indivisible function. A consequence of this is that in passing from the normal to the pathological state the psychical, motor, sensitive and trophic troubles may all be associated; this is proven in all the affections which originate in insufficient or abnormal development of nervous centers, as chorea, tic, tabes, syringomyelia, muscular atrophy and general paralysis. Coëxistence of motor and psychical trouble is not characteristic of any one malady, but a fact only wanting confirmation, whether in neuropathology one studies the diverse affections of the neuromuscular system which explains degeneration, or whether in psychiatry one examines the various psychoses.

For instance, in Huntington's disease mental troubles more or less pronounced will invariably coëxist with motor affections. This affection is not confined to the one member of the family affected, but often extends to more members, either in motor or mental degeneration. Facts bearing on this relationship of psychical and motor trouble, may be observed in Thomsen's disease, and Parkinson's disease may also be cited, where this point has not been sufficiently noticed. In a family in which Parkinson's disease exists there may be found not only other members with the same disease, but also choreaics, hysterics, epileptics and aliens, with cases of ataxia and headache; and there is especially emphasized the coëxistence of mental trouble, hallucinations, illusions and disorder of general sensibility with Parkinson's disease. Further, the same psychomotor complex is observed when, instead of considering muscular troubles, the psychoses are studied. General paralysis is put aside, as hallucinations, illusions and delirium constitute only a complementary syndrome, as mental trouble in chorea. In melancholia, however, besides troubles of sensibility, hyperesthesia, anesthesia and dysesthesia, which are prominent, functional muscle troubles are often seen. This is still more noticeable in certain forms of mental confusion, especially in *démence précoce,* whether by automatic movement or by that curious syndrome known as katatonia.

One case is reported to show the coëxistence of motor and mental degeneration in the same patient. A man of twenty-six years was afflicted with muscular atrophy. A summary examination sufficed to show that the first segment of the limbs was more affected than the extremities. The thigh was most atrophied, the leg notably so, while the foot showed only deformation due to tendinous retractions; corresponding gradation showed in the upper limbs, where the hands were almost

normal. After a more methodical examination all the muscles of the shoulders were found to be atrophied. The deltoid was gone, the pectorals, the rhomboideus were scarcely indicated, and wide detachment of the thoracic bone was easy. The circumference in the middle of the upper arm was only 14 cm., and the lower arm was larger. Pronation movements were easy, but supination limited. The tendinous retraction complications of the lower limbs determined a special attitude: the thigh, when abducted, was turned out; the knees, moved one from the other, rested on the bed; the legs were bent at right angles with the thighs and immobilized in this position by tendinous retractions; the heels were near together; the foot extended, forced by the shortening of the tendon of Achilles. The trapezius, the muscles of the vertebral grooves, and even those of the abdomen, were affected. The patient had a very large lower lip; the eyes could hardly be closed, but the lids were raised at the least effort. There was marked orbicular weakness. A smile stretched the mouth out of all proportion, and at the same time vertical creases appeared about the commissures of the lips. There was no deviation of the vertebral column. Thus the patient showed some physical stigmata of degeneration, asymmetrical face, irregular teeth, pointed arch and adherent lobule of the ear. It could hardly be doubted that this was incipient progressive myopathy, although the fibrillary contractions so often seen with this were not present. In the electrical examination of the muscles many had normal reaction, but somewhat weakened. In some the reaction of degeneration occurred.

Up to fifteen years of age development had been normal; at that time the wheel of a heavy wagon had passed over the patient's trunk, but there was no loss of consciousness, or any alarming symptoms, and he was able to walk away. Yet this accident was responsible for the beginning of his affection, in spite of the long time elapsing until the beginning of the muscular weakness. Six months after a cane was necessary for walking, and five or six years after, crutches. He had been in bed three years at the time of reporting.

This primitive progressive myopathy is usually an inherited disease, but not so in this case. At this point the members of the family had a strange idea that the forced chastity of invalidism was aggravating his condition, and after mature deliberation they put him in a coach and took him on a round of the houses of prostitution, bringing him home very excited, delirious and probably intoxicated. This continued the following days, with hallucinations of sight and hearing. There probably was some mental trouble before this, as it could scarcely have begun so suddenly after this singular excursion. However it be, from that moment delirium was incessant, various hallucinations of riches, persecution, alternating excitement and depression. There were frequent attempts at suicide. This mental state grew worse; at the beginning one could fix the patient's attention and obtain a response, which was later impossible. He talked incessantly and incoherently, simply starting on ideas without finishing any thought. There was no trouble with the iris, and there could not be general paralysis. The cephalorachidian liquid showed no figured elements. From the psychical point of view it was then to precocious dementia that the mental troubles might be traced. This was not the first case of the coëxistence of these troubles, and the article cites many others, with comments from a more modern standpoint.

He concludes that if the purely motor nerves are affected, there will be motor trouble as in the disease of Sydenham, of Huntington's chorea, or in tic. If more specially trophic nerves are affected, there will be muscular lesions, as in Thomsen's disease, or primitive progressive my-

opathy or Parkinson's disease. Finally, if other nerves are affected there may be similar psychical troubles, as in chorea, myopathy and Parkinson's disease. Parkinson's disease is a distinctly degenerative malady; there are not only atrophied muscular fibers, but hypertrophied fibers similar to those of myopathy, so that it is placed both by pathological and clinical anatomy in this class.

In conclusion, all of these diseases mentioned with the mental troubles accompanying them, are but diverse manifestations of degeneration, forming a natural group, which the author calls musculo-psychic, of conceptional or hereditary origin; or, another name, *myopsychic* diseases. JELLIFFE.

Neurologisches Centralblatt.

(1902. April 16, No. 8.)

1. Short Communication regarding a New Staining Method for the Central Nervous System. H. VON SCHRÖTTER.
2. A Case of Autochthonous Brain Sinus Thrombosis. DR. GOOD.
3. Further Concerning Asthenic Paralysis with Autopsy (E. Flatau). S. GOLDFLAM,

1. *New Staining Method.*—The use of the Alizarian group of stains, *i.e.*, blue, green, yellow, orange, is applied to nervous tissues with excellent results. A ½ per cent. solution is used for twenty-four hours; the tissues are then differentiated in running (tap) water, treated with obsolute alcohol, etc. This gives a diffuse tissue stain, bringing out all the elements of the nerve tissue.

By using a 3 per cent. solution of the Alizarian solfonal and salt of sodium, and adding a drop of a 5 per cent. oxalic acid solution, orange-yellow color is produced. If the tissue is stained in this and then heated by a 3 per cent. soda solution after washing in distilled water, a good selective stain for the nerve sheath results.

2. *Brain Sinus Thrombosis.*—Good reports a case of extensive sinus thrombosis for which no determining factor could be discovered. After a careful study of the literature he concludes that such cases are not diagnosable.

3. To be continued.

McCARTHY (Philadelphia.)

Neurologisches Centralblatt.

(1902. April 1, No. 7.)

1. Judges and Expert Evidence. H. HOCHE.
2. Cacodylic Acid Therapy. H. SMIDT.
3. Hysteria Problems. RANKE.
4. Asthenic Paralysis, with Autopsy. S. GOLDFLAM.

1. *Expert Evidence.*—A discussion as to the value of a knowledge of medical questions by the Judge, in cases where the expert testimony is indefinite or inconclusive.

2. *Cacodylic Acid Therapy.*—The literature of cacodylic acid as a therapeutic agent in nervous diseases. It has been found of benefit in the following diseases: Neurasthenia, chorea, paralysis agitans, psychoses, etc. Hypodermic use of the drug is recommended, to avoid gastric complications.

3. *Hysteria.*—A controversial paper as to the nature of hysteria and

as to what symptoms are to be considered essential for the diagnosis. It is an answer to a paper on a similar subject by Nissl.

4. To be continued.

McCarthy (Philadelphia).

MISCELLANY.

Hyoscine in Paralysis Agitans. Dr. Judson S. Bury (Lancet, April 19, 1902).

The author says that hyoscine is probably the most useful drug that has hitherto been tried in the treatment of paralysis agitans. As a rule it diminishes or arrests the tremor, checks the troublesome restlessness and the desire to change position, and relieves the "hot flushes" and unpleasant sensations of heat by which these patients are often troubled. It is important to remember, however, that hyoscine is a powerful drug, and one which must be administered with great care. Merck's hyoscine hydrobromate is probably the best preparation, and it is safer to give it by the mouth than to inject it. Quoting from Williamson, he says, "A prescription which is useful is one-eighth of a grain of hyoscine hydrobromate in six ounces of chloroform water. At first two teaspoonfuls of this may be given, then three, four, or five teaspoonfuls. If necessary the dose may be increased to six teaspoonfuls (1-64th of a grain), providing toxic symptoms are not produced. The hyoscine is best given in the morning, just after breakfast, and again in the evening, just before going to bed, if the patient is troubled with restlessness and sleeplessness during the night."

Certain Clinical Types of Brain Syphilis. Pearce Bailey (Medical Record, June 21, 1902).

The most important clinical groups of brain syphilis are the apoplectic, the meningitic, those characterized by isolated oculomotor palsies, and those characterized by somnolence; also those which correspond with the anatomical types of gummata and cerebro-spinal affections. Acute specific meningitis may be suddenly changed by the addition of apoplexy. These acute apoplectic seizures of cerebral syphilis are rather common. The writer believes that cutaneous disturbances of sensibility have been rather more common than usually mentioned in the text-books, and disturbances of the higher visual tracts distinctly more so; this includes especially bilateral homonymous hemianopsia, which the writer finds frequently of syphilitic origin. Focal signs may be wholly absent in some vascular cases. Often the symptoms of brain syphilis are of gradual, instead of sudden onset. At first they are chiefly mental, the patient becoming irritable, inattentive and forgetful, headache is not prominent, and disturbances in the special brain symptoms are often late in appearing. Somnolence is a frequent and characteristic sign of brain syphilis, improving with the other clinical symptoms, or deepening into coma, as the disease increases. Cases occur characterized by attacks, including those of an epileptoid character, usually focal or Jacksonian, with numerous associated symptoms, such as aphasia, anesthesias, vertigo, or mental symptoms. Disease in the blood vessels usually is found in these patients. General epileptic attacks, or "late epilepsy," are common in brain syphilis. A combination of trauma and syphilis is said to produce a variety of serious conditions. The writer believes that trauma, as a cause of cerebral syphilis, is rare. Syphilis, in its attacks on the central nervous system, has the tendency to make deposits, sometimes microscopic, in both brain and spinal cord. Clinically, the cere-

bro-spinal character may or may not be evident. Some cases seem to be cerebral, some spinal.

The relation of psychoses to syphilis is obscure. Maniacal and melancholic mental states, or dulness and sleepiness, merging into dementia, are ordinary clinical expressions of organic brain syphilis. Can these be distinguished from functional cases? The writer believes that there are a few cases in literature which indicate that a diagnosis of syphilis can be made in these cases, and a direct antisyphilitic treatment give benefit. Post-syphilitic dementia is in all probability due to vascular disease. In most cases of syphilitic mania, meningitis, or vascular disease, could be demonstrated. W. B. Noyes (New York).

Dormiol in Epilepsy. J. Hoppe (Münchener med. Wochenschrift, April 29, 1902).

Dormiol has been used in eleven cases of status epilepticus, and it has been found that, so far as efficiency is concerned, it ranks equally with amylene hydrate, and has not any after-effects. The administration was per rectum in the form of two to three tablespoonfuls of a stock solution, 10 to 150, in from one-quarter to one-third liter of water. For continued use in epilepsy, dormiol is no more suited than is chloral or amylene hydrate, except, perhaps, in certain cases of epilepsia nocturna. In cases of epileptic confusion, with motor restlessness, it failed completely. Jelliffe.

Phototherapy and Neurasthenia. P. Joire (La Semaine Médicale, 1902, No. 17).

The important modifications which one notes in the nervous system following the use of colored rays of light, or of decomposed light, induced this author to employ phototherapy in treating neurasthenic troubles. In his experience the red rays are especially to be recommended for this purpose, not only because of the sedative effects which they appear to produce upon the nervous system, but also because of their considerable power of penetration and of regulation exercised upon the blood circulation. The treatment by red light is particularly efficacious in cases of hyperesthesia, which constitutes, as is well known, the point of departure of most of the characteristic symptoms of neurasthenia, such as headache, vertigo, pain in the back, etc. In cases in which one sees, after the disappearance of painful symptoms, a certain degree of cerebral depression, it will be well to employ light cures of other orders, and to make, according to the state of the patient, a variation of color and intensity and manner of application. The calming action of phototherapy, and especially of exposure to red rays, seems also to have the power of increasing the strength of the patient, augmenting the appetite, regulating digestion, and governing general nutrition. In spite of their great susceptibility, neurasthenics undergo treatment with red light without difficulty, perhaps because the vibrations of this color are comparatively slow. Jelliffe.

Multiple Neuritis. Daniel R. Brower (Medical Record, June 21, 1902).

The disease is the result of one kind of poison, and while the action of this agent may be limited to the peripheral portion of the neurone, yet there are cases in which the cell-body as well is impaired. The principal poisons producing multiple peripheral neuritis, are alcohol, lead, arsenic, and the toxine of various micro-organisms, especially those that produce diphtheria, influenza, typhoid fever, gonorrhea, syphilis,

leprosy, and beri-beri. In the typical cases there is a symmetrical localization of motor, sensory and vasomotor symptoms. In earlier stages the symptoms are the result of irritation of the nerve, in the later of destruction. Different cases vary in developing one set of these symptoms, lead paralysis, Landry's paralysis and tetany being distinctly motor; pseudo-tabes and leprosy being sensory; and erythromelagia and Raynaud's disease being vasomotor in clinical type. The commonest form of multiple neuritis arises from alcoholism, and ordinarily presents a mixed type. Its diagnosis is based upon such evidences of chronic alcoholism as indicated by digestive, circulatory and nervous systems. The onset is insidious; the earlier symptoms are numbness and tingling, cramps and tremors, with painful symptoms and cutaneous anesthesia a little later, and sometimes a muscular hyperesthesia. The paralysis soon results in quadriplegia, with toe and wrist-drop, sometimes there is at first an exaggeration of the knee-jerk, followed by a loss of knee-jerk. Progressive atrophy occurs. Marked psychical disturbance sometimes occurs, due to the alcoholism.

W. B. Noyes (New York).

Clinical Differentiation of Brain Syphilis and General Paresis. W. A. M'Corn (Brooklyn Medical Journal, February, 1902).

The writer reports a case with the following symptoms: A capable business man, after a week of general apathy, headache, and insomnia, becomes incoherent, irrational and appears greatly confused and stupid. The muscular coördination becomes so marked that it is nearly impossible for him to walk, talk or care for himself in any way. He had had syphilis three years before. Examination of pupils was negative, the knee-jerks exaggerated, no fibrillary tremor of the tongue or face; facial expression dull and apathetic. Romberg symptom extreme. No grandiose ideas, memory poor for recent events. Owing to his unsteadiness on his feet he was put to bed and treated antisyphilitically. The patient improved for a short time. Three months after admission a divergent strabismus of the right eye, with ptosis, developed. Almost simultaneously the patient became somewhat filthy, mentally dull, and stupid. The diagnosis of paresis was made by eminent specialists, but is disputed by the writer, for the following reasons: Brain syphilis follows infection closely, from one year to ten. Paresis has a remoter syphilitic origin, and seems to be more the result of a parasyphilitic toxin. Mental stress is as essential a factor in paresis as heredity is insignificant. The course of the two affections shows a marked difference. In paresis it is slowly, but regularly progressive, the course of brain syphilis is extremely erratic. A degree of dementia out of all proportion to the duration of the disease is often followed by sudden and rapid improvement, to again lapse into a stupid, apathetic state similar to that found in the last stage of paresis. There is a wide difference in the mental symptoms, other than the intellectual defect. Paretics, as a rule, have a marked euphoria from the onset of the disease, accompanied by absurdly extravagant delusions, while the syphilitic is more depressed, and has hypochondriacal, melancholic delusions.

The physical symptoms of syphilis point almost invariably to focal and more or less limited lesions, while in paresis, to a diffuse involvement. General, tremulous and ataxic movements are not common to syphilis; the speech disturbances are not pronounced, and the headache in syphilis is persistent, diffuse, worse at night; while in paresis it is usually paroxysmal and localized. Optic neuritis is common in syphilis.

W. B. Noyes (New York).

Book Reviews

Die Physiologie und Pathologie der Coördination. Eine Analyse der Bewegungsstörungen bei den Erkrankungen des Centralnervensystems und ihre rationelle Therapie. Von Dr. Otfrid Foerster, Assistent der Psychiatrischen Klinik der Universität Breslau. Gustav Fischer, Jena. G. E. Stechert, New York.

We believe this to be the first attempt at a monographic treatment of that most interesting of physiological functions, coördination, and of its pathological corollary, incoördination. Surely the importance of this subject has merited its separate treatment long before this.

The separate phases of incoördination, in varying nerve affections, have been but illy analyzed or brought into relation with each other. The present most excellent volume is worthy of the warmest praise. It is not only a credit to its author, as an evidence of his scientific acumen and energy, but is intrinsically of great value to the practitioner in all fields of activity, especially in those in which questions of neuro-muscular functions are involved.

The author first considers Physiology of Coördination, considering coördination of the muscles and their strength and the coördinating functions of the central nervous system; spinal, cerebral, and cerebellar coördination, and compound, or mixtures of these. In a separate chapter the general pathology of coördination is discussed.

The major portion (78-305) of the pages is taken up with a most careful and thorough study of coördination in tabes dorsalis, in which the entire musculature and its activities are utilized in affording abundant important facts.

The work is of great merit, even if largely taken up with the study of one diseased state, and will be welcomed by all students of neurology.

Jelliffe.

The Drug Habits and their Treatment. By T. D. Crothers, M.D., Superintendent Walnut Lodge Hospital, Hartford, Conn. G. P. Engelhard & Company, Chicago.

Coming as does this small brochure from the pen of an observer as well as literary man, the result is both of practical value and of readable merit.

Dr. Crothers treats, in a very simple manner, the alcohol habit, dipsomania, opium inebriety, and cocaine and other drug habits. Altogether there are but some 90 pages in this small volume, but there is much food for reflection contained therein. Jelliffe.

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Original Articles.

A CONTRIBUTION TO THE RADICAL CURE OF EXOPHTHALMIC GOITER WITH THE ULTIMATE RESULTS IN EIGHT CASES TREATED BY THYROIDECTOMY.*

By J. Arthur Booth, M.D.,
New York.

In a paper entitled "The Thyroid Theory in Graves' Disease," read before this Association, May 30, 1894, and in a second communication of May 30, 1898, the following conclusions were presented: (1) Cases of Graves' disease may be entirely cured by operative measures; (2) pathological and clinical evidence is in support of the view, that the symptom-complex is the expression of a primary neurosis, multiplied by a secondary glandular intoxication; (3) while the ultimate cause of the disease of the gland is still a matter of speculation, and a mortality of more than seven per cent. after operation, is reported, we cannot recommend operative interference as a routine plan of treatment; (4) sudden death may occur in the course of, or soon after, operation, and has not as yet received a satisfactory explanation.

Investigations during the past five years have not resulted in causing any material change in the above conclusions. Still

*Read at the annual meeting of the American Neurological Association, June 5, 6 and 7, 1902.

a study of the large number of cases treated surgically, and of the data derived from their observation for long periods after operation, as well as the results of operative measures other than thyroidectomy, have necessitated corresponding modifications in my views.

Continued and general interest has been excited in the problem of solving the pathogenesis of so curious a symptom-complex, recognized under three names, having three diagnostic symptoms, for which, in explanation, three theories have been advanced, and three surgical measures recommended for its cure; and although the time is not yet ripe for accepting any exclusive theory of the causation of the disease, nevertheless there seems to be more of an approximation of all.

In the attempt to explain the different grouping of symptoms, three classifications may be adopted: (1) those cases dependent on changes in the central nervous system; (2) those due to disease of the cervical sympathetic; (3) those arising from excessive or altered function of the thyroid gland.

In the first class of cases, there are distinct lesions in the medulla, which have been verified by a number of autopsies. Another reason for assuming that the origin of the symptoms is in the central nervous system, is the evidence derived from clinical observation, such as (a) the predominance of the disturbances of the vagus at its central origin in the medulla, as demonstrated by the excessive tachycardia; (b) the occurrence of cases in which there is no goiter and no evidence (operative or by autopsy) of disease of the cervical sympathetic; and finally (c) the frequency of sudden death attributable to minute hemorrhages. In this group of cases no benefit is to be derived from operative measures.

The second class of cases is characterized chiefly by symptoms referable to the sympathetic system; abundant evidence of this being furnished both by cases and observations. The results of a number of autopsies have also shown lesions of the sympathetic, and especially of the inferior cervical ganglia; the usual change found being a thickening, which may be unilateral or bilateral. In conclusion, most important in support of this view, are the numerous recoveries recently reported from oper-

ations on the sympathetic. Rehn, of Frankfort, in 32 cases of resection of the sympathetic, reports as follows: 28 1-10 per cent. as cured, 50 per cent. improved, 12 1-2 per cent. unimproved, and 9 3-10 per cent. deaths. Jennesco, in 15 cases, 6 were cured, 4 improved; in 5 sufficient time had not elapsed to give results. There were no deaths.

Under the third, and last classification of cases, it now remains for us to consider those arising from excessive or altered function of the thyroid gland. In this class we have positive proof of changes in the thyroid in a very large number of instances. This fact is so well known and acknowledged by all that it does not seem necessary to do more than refer briefly to this part of the subject. We find enlargement of the gland, involving the whole glandular tissue, with the vascular structures greatly in excess. Certainly, I know of no case in which the gland has been found normal after death. Our main reasons, then, for assuming that these cases have their origin in the thyroid are (a) the study of clinical evidence; (b) the results of experimentation; (c) post-mortem findings; (d) results of operative measures.

Most important in support of this view are the records of Kocher, Schultze, Kummeil, Mikulicz, Rehn, Moebius, Jabouley, Jennesco, and others. Schultze and Rehn report that in 319 cases, 175 were cured (51 per cent.), 89 improved (28 per cent.), 13 unimproved (4 per cent.), 41 died (13 per cent.).

Rehn, of Frankfort, regards the thyrogenesis of the disease as proven, and states that the thyroid gland dominates the disease and that it is always changed even if this is not visible externally.

Renbach, of Breslau, reports the following results from the clinic of Mikulicz; of 18 patients treated exclusively by internal medication for a year or more, 17 recovered from the operation, 1 died of secondary hemorrhage. The cure was permanent in 10 cases, 8 of which had been observed 4 years. One patient who had been given up by a physician was cured. Of the seven remaining patients, all were materially improved. Kummel, of Hamburg, gives the following results in 177 patients; 57, 6 per cent. cured; 26, 5 per cent. improved; 2, 3 per

cent. unchangel; 13, 6 per cent. died. Certainly results such as these prove conclusively that in a large proportion of patients the thyroid is the pathological factor. As additional evidence, I now wish to report the present condition of seven cases that have been under observation for some time.

Case I.—The patient, Louise E——, aged twenty-four years, was first seen January 23, 1893. The family history is entirely negative. She never had any illness worthy of note up to the date of the present trouble. About two years ago she first noticed enlargement of the neck, and three months later the eyes became affected. The patient is positive that there were no heart symptoms until six months later, when palpitation, throbbing in the vessels of the neck, shortness of breath, and flushing of the face appeared. All these symptoms gradually increased in severity, and in addition there was marked insomnia, because of the tumultuous action of the heart. Both eyes are very prominent, especially the left, and the lids do not follow the movements of the eyeballs. The pupils are moderately dilated, reacting to light and accommodation. Vision not impaired and fundus normal.

The enlargement of the thyroid is marked, the right lobe being the larger. On inspection, pulsation is quite noticeable over this region along the sides of the neck. Over the middle of the thyroid the neck measures fourteen inches. The pulse is 150 and of high tension. The apex beat is diffused, but no murmur is present. Respirations, 24 to the minute. Measurement over bust thirty-one and one-half inches, and on full inspiration thirty-two and one-fourth inches. During a period of six months, from January to July, 1893, the patient received daily applications of galvanism and appropriate internal medication. At the end of the above period there was some improvement in all the symptoms, including a slight reduction in the size of the neck, and the eyes were less prominent. During July, August, and September, the patient received no regular treatment, but faithfully carried out my instructions as to resting several hours each day. After this time she relapsed into her former condition; the exophthalmos was well marked, and the right lobe of the thyroid very prominent. Further treatment failed to make any material alteration in her appearance, or to cure the many subjective symptoms from which she suffered; so operative interference was considered and decided upon.

The operation was performed by Dr. B. F. Curtis on November 11, 1893. A right transverse incision was made just below the hyoid bone and a vertical incision from the inner

end of this at the median line. The flap thus made was turned back and exposed the right lobe of the thyroid. There was but little bleeding, the vessels being carefully dissected out and tied as found. A supernumerary lobe was found attached to the inferior angle and pressing directly upon the trachea; this was removed. The entire exposed mass was then separated from its bed and from the trachea, the isthmus ligated, and the whole right lobe removed.

During the operation the pulse was between 180 and 200, irregular and intermittent, but respiration continued normal and no stimulation was necessary. By evening she had made a good recovery from the ether, complaining of severe pain in the throat. Voice not husky and of good strength. Up to the fourth day after the operation, nothing occurred worthy of note; the patient then had much nausea and vomiting, accompanied by a sharp rise in temperature. These symptoms continued for two days and then rapidly disappeared. On the tenth day the dressing was removed for the first time, and the wound was found healed by primary union.

The course of the case after this was satisfactory, and on the sixteenth day she was discharged from the hospital.

Inspection of the specimen after removal did not reveal any great degree of vascularity; it was homogeneous in appearance and hard in consistence. Microscopically, the tissue showed the structure of normal thyroid gland with the alveoli moderately distended with hyaline material, and in places thickly crowded small cells, both in the alveoli and in the stroma, but principally in the latter.

May 30, 1894, seven months since the operation, there has been a progressive and decided improvement in all the symptoms; especially is this so with regard to the various nervous phenomena, viz.: insomnia, restlessness, flushing of the face, excessive sweating, and palpitation. The eyes are less prominent, and the pulse now varies between 96 and 110, as compared to the former ratio of 120 to 160. The appended table shows the pulse average for nine months:

AVERAGE OF PULSE RATE:

August, 1893, 148; September, 1893, 156; October, 1893, 136; November, 1893, 146; December, 1893, 120; January, 1894, 120; February, 1894, 110; March 1894, 104; April, 1894, 104.

For the past six years she has been employed in one of the large department stores, being on her feet most of the time from eight o'clock in the morning until six at night, not missing a day

from her work, and during this period she has not been disturbed by any of her former symptoms.

An examination made on this date (8½ years since the operation), shows that the prominence of the eyes is still present, but to a much less degree; the wide staring expression has entirely disappeared. The neck, formerly 14¼ inches, now measures only 13 inches. The tremor has disappeared also. She states that she has no palpitation, dyspnea, or excessive sweating; nor is she troubled with insomnia. The pulse is 80. All of the above symptoms were present to a marked degree before the operation, so that from her present condition I think we are justified in believing her cured.

Case II.—Anna D——, aged twenty years, single; seen April 23, 1893. Three years before she noticed enlargement of the neck and three months later a prominence of both eyes. She became very nervous, could not sleep, and was very much disturbed by frequent attacks of palpitation. The right lobe of the thyroid was removed on May 11, 1893. Marked improvement in all the symptoms followed. One year after the operation there was entire absence of any exophthalmos; the pulse, formerly 140, was down to 100, and the nervousness had disappeared. On April 26, 1902, nearly nine years since the operation, her condition was satisfactory; there was no prominence of the eyes, and the pulse was 82.

Case III.—Nettie D——, aged forty-two years, single, was seen June 22, 1894, and the following history obtained: Twelve years ago the patient received a severe fright, the nature of which she will not give. She was very much prostrated and became very nervous. Six months later she noticed a bunch on the right side of the neck, which gradually increased in size and finally extended over to the other side. Two years ago she became very much annoyed by frequent attacks of palpitation and shortness of breath after slight exertion, also by a throbbing in the vessels of the neck. Within the last year the eyes have become very prominent. Recently she has passed large quantities of urine, and has been under treatment for diabetes.

Examination revealed all the symptoms of the disease to an extreme degree. She was admitted to St. Luke's Hospital on September 24, 1894, and, no kidney complications being found, the operation of partial thyroidectomy was performed by Dr. Curtis on October 1, 1894. Shortly after the effects of the ether had passed off there developed a high degree of fever; the patient rapidly passed into a comatose condition and finally died twenty-four hours after the operation. An examination of the urine revealed a large percentage of albumin, hyaline and granular casts. No autopsy was made, and therefore, it is difficult

to arrive at any conclusion as to the real cause of death; but from the examination of the urine and the symptoms it is probable that the final condition was one of uremia and not to be explained by the sudden absorption of an excess of the secretion of the thyroid gland.

Case IV.—Nellie C——, aged seventeen years, single; seen November 5, 1895. When six years old, a sister, taking her in her arms, made a pretence of throwing her out of the window. She was very much frightened, and an attack of what was called brain fever followed. She was confined to the bed for several months, and during this time had a number of convulsions, but finally made a good recovery. She remained well until the appearance of menstruation six years later; about this time, when thirteen years old, a swelling of the upper lids of both eyes was noticed, which has gradually increased and now has become so noticeable as at once to attract attention. This symptom is more marked in winter than in summer.

She now complains of frequent attacks of palpitation of the heart, accompanied by throbbing in the neck and profuse perspiration; also of general nervousness and occasional headache. She has never noticed any prominence of eyes, enlargement of the neck, or swelling of the hands or feet. On examination the patient presents the appearance of a case of Graves' disease. When we examine the eyes, however, no exophthalmos is discovered, but instead a very marked and peculiar edema of both upper lids. It is not a true edema; no pitting follows pressure, and it does not cause the closure of the lids, such as is produced by ordinary edema. Movements of the eyes and lids are harmonious. On inspection there is no decided prominence of the thyroid gland, but on palpation swelling and a diffused hardness of both lobes are made out. The heart action is agitated and pulse rate rapid, averaging 120 beats to the minute. With the exception of loud hemic murmurs at the base, the heart is normal. Face, neck, and both hands are covered with beads of perspiration. There is a slight tremor of the fingers.

One month later (December 5th), prominence of the left eye appeared. Having been unable to do any work for some time, even to attending to minor household duties, the patient willingly consented to operative interference, and on December 15th she was admitted to St. Luke's Hospital, where a few days later the right lobe of the thyroid was removed by Dr. B. F. Curtis. Her recovery from the effects of the operation was rapid and the progress of the case up to the present time has been entirely satisfactory. It is now seven years since the operation, and during this time there has been a complete disappearance of all nervousness; the throbbing and palpitation have ceased, and

with two exceptions the pulse has not been above 90, most of the time varying between 80 and 86; no exophthalmos. Although the improvement in the symptoms just mentioned has been marked, the peculiar edematous swelling of the eyelids still persists.

Case V.—Amelia B——, aged twenty-one years, single, domestic; seen with Dr. Curtis November 29, 1895. Present trouble began four months before, when she felt a movable, painful swelling, about the size of a cherry, on the left side of the throat, which caused her pain on swallowing or talking. About a month later it had grown to about the size of a pigeon's egg and was quite visible. She became very nervous, could not sleep, and had attacks of palpitation. Examination did not reveal any prominence of the eyes. Pulse rate, 110 and irregular.

The left lobe of the thyroid gland was removed; the patient made a good recovery from the operation and was discharged from the hospital on December 2, entirely relieved of the pressure symptoms from which she suffered. Three months later the nervous symptoms had disappeared, the pulse became normal, and when seen on May 18 last, she was perfectly well, the pulse being 74; no nervousness or insomnia.

During the past two years no examination has been made, but up to that time there had been no return of the symptoms of which she had complained.

Case VI.—Addie T——, aged thirty-five, married. This patient was referred to me by Dr. Curtis on January 10, 1896. Her general health was good up to ten years ago; then she first noticed a swelling on the left side of the neck, which gradually increased in size. Palpitation of the heart and general nervousness appeared five years later. At no time had she noticed prominence of the eyes. Examination revealed a decided enlargement of the left lobe of the thyroid, throbbing along the vessels of the neck, and a pulse of 120. The eyes appear normal both in size and movement.

One year ago her sister died with symptoms of the disease, and now another sister has the same trouble.

On January 11, 1896, partial thyroidectomy was performed by Dr. Curtis, the left lobe being removed. No shock followed, and she made an uneventful recovery from the operation. Two years later all nervous symptoms had disappeared, there had been no tachycardia and the pulse was 84.

Case VII.—Mabel C——, aged twenty years, single. This patient presented all the symptoms of the disease to a marked degree and when I saw her on November 20, 1896, her condition was a miserable one. Enlargement of the neck was first noticed one year previous, which was followed by general ner-

vousness, tremor of hands, excessive sweating, attacks of dyspnea and palpitation. She complained also of frequent attacks of diarrhea. The pulse rate was very rapid, 160 to 180. Both eyes were prominent. After six months' rest in bed, and having received no benefit from either this or many therapeutic measures, she was prepared for operation, and on June 28, 1896, the entire right lobe of the thyroid gland was removed by Dr. Curtis. She returned home February 19, 1897, unimproved. Since then she has certainly been in better condition, and when I saw her on May 8, 1902, the eyes were not so prominent, the remaining lobe of the thyroid was smaller, the nervousness was less; she was seldom disturbed by palpitation, and the pulse was 100.

Case VIII.—Mary M——, aged thirty-six years, single; bookkeeper. Patient referred to me by Dr. David Webster on October 12, 1897. Onset of symptoms one year previous, consisting of general nervousness, prominence of eyes, rapid pulse —128 to 130—tremor of hands, and enlargement of neck. Admitted to St. Luke's Hospital on October 28, and on November 13, 1897, the right lobe of the thyroid excised by Dr. Curtis. Her recovery from the effects of the operation was uneventful, and she returned to her occupation as bookkeeper in five weeks, feeling better. When seen on April 26, 1902, she stated she had been very much improved in every way. She is less nervous, sleeps as well as ever. She is now able to write and keep her books neat; formerly she was not able to do this on account of the tremor of the hands, which was so marked as to resemble that of chronic alcoholism. She has hardly any palpitation now, and the pulse varies from 80 to 90. Prominence of eyes hardly noticeable.

It will be seen from the preceding that of the eight cases, six were permanently cured, one improved and one died.

The cause of sudden death, after or during the operation, is somewhat difficult of explanation, but to some extent may be attributed to a faulty technic in removing the vascular goiter, as well as to the reduced vitality of the patient, whose heart has been weakened; or to renal complications. The theory that it may be due to the absorption of the thyroid juice does not now seem tenable, owing to the care at present exercised in preventing this in the removal of the diseased part. In the third case, in which death occurred forty-eight hours after recovery from the effects of the ether, there was a history of an attack of diabetes and a specimen of urine obtained while the patient was in a

comatose condition, showed a large quantity of albumin and a variety of casts.

The writer acknowledges the difficulty of separating and placing the individual case in one of the given groups, in order to select the proper operative measure; especially is this so at this time when all is as yet so obscure concerning the true pathology of the disease. Still, with greater care in considering the onset and course of the disease, giving due weight to the hereditary element, if present, and finally noting which of the symptoms first appeared; the grouping suggested may prove not merely of theoretical, but also of practical assistance. In seven of the eight cases, the enlargement of the thyroid was the first symptom to appear, and the satisfactory results obtained in six were due in all probability to the operative measures upon the gland itself.

In another class of patients there is no enlargement of the gland, and in these one should consider the advisability of a resection of the sympathetic. Since the introduction of bilateral resection of the sympathetic, it has been performed with sufficient frequency to permit a comparison of its results with those of thyroidectomy, and from the reports of cases thus far published, we are led to conclude: that, with the latter, sudden deaths are more numerous, and the relief from the exophthalmos less common. In the eight cases just reported, exophthalmos was present in six. After operation it disappeared in three and was improved in three. Although the results accomplished in my cases have been so satisfactory, I am not prepared to state that the treatment by operative measures is the best and most efficient; but it seems to me, that more marked and rapid benefit has been obtained under surgical than medicinal treatment. Hence the former must receive due consideration, especially after we have convinced ourselves of the futility of other measures.

If the case does not improve under the customary treatment, too much time should not be allowed to elapse before resorting to the knife, for too prolonged a delay materially increases the risks of the operation and renders success less certain and permanent.

Reviewing all these facts, then, we may formulate the following conclusions:

(1) Cases of Graves' disease may be completely cured both by thyroidectomy and bilateral section of the sympathetic.

(2) In view of the fact that some cases are cured by internal medication, there must be a certain proportion in which the affection does not induce structural changes in any organ.

(3) No theory can be regarded as adequate without taking into consideration the function of the thyroid gland.

(4) Three factors must be considered in the production of the disease: (a) The central nervous system; (b) the connecting fibers: sympathetic and vagus; (c) the thyroid gland.

(5) A lesion of one of these parts may produce a specific alteration in the others, the consequences of which, together with the exciting cause, may give rise to the symptoms of Graves' disease.

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ACUTE HEMORRHAGIC CORTICAL ENCEPHALITIS.[1]

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The study of the acute non-purulent inflammation of the gray matter of the brain has received much attention during the last decade especially, but so much space and time has been devoted to the subject of the acute hemorrhagic inflammation of the gray matter surrounding the floor of the fourth ventricle, and especially the canal of Sylvius, that the other, far more important study of cortical affections of this character, has been pushed into the background. This in a large measure is, and was, due to the striking picture presented by Wernicke's acute encephalitis hæmorrhagica superior, whereas the cortical form is either frequently mistaken on the one hand for leptomeningitis, acute mania or, occurring as it so frequently does, intercurrent or after an acute infectious disease, is either overlooked or taken for hemorrhage or softening. You will easily recognize the importance of properly diagnosing this disease, not only for a scientific reason, but also because in favorable cases prompt and, in my experience, continuous treatment may effectually remove the pathological condition, and in a measure prevent the remote consequences.

This variety of encephalitis is called by the German authors The Strümpell-Leichtenstern form. Strümpell[2] first directed the attention of the medical world again to this subject by his paper on infantile cerebral palsies, as being frequently caused by encephalitis, and many years before this (1865) Virchow[3] had made exhaustive studies of the pathology of encephalitis of the new born.

In 1885 Leichtenstern described a case of acute non-purulent encephalitis occurring during the course of an epidemic of cerebro-spinal meningitis, and he was the first to call up the thought that the disease was due to an infection. The influenza

[1]Read at the annual meeting of the American Neurological Association, June 5, 6 and 7, 1902.

epidemic of 1889-1891 brought out typical cases. Those of Fürbringer[5] especially, Koenigsdorf[6] and Schmidt[7] deserving special mention.

After the bacteriological origin had been denied by Strümpell, Brickler, Goldscheider and others, although in the typhoid, diphtheritic and other zymotic diseases, streptococci had been found, Pfhul, and especially Nauwerk, by finding Pfeifer's influenza bacillus, were enabled to furnish the proof that the disease was due at times at least to direct bacterial invasion of the brain and not to ptomaine or leucomaine intoxication or to secondary infection.

Oppenheim's monograph on encephalitis in the Nothnagel series, to which I am indebted for most of the above historical data, contains a complete bibliography on the subject, up to 1897.

The etiology of acute hemorrhagic encephalitis of the cortex is usually directly or indirectly bacteriological or zymotic. Either the inflammation is due to the direct invasion of the cortex of the brain by the bacteria which cause the primary disease, as for instance in influenza, where we have positive evidence, or in diphtheria and typhoid, where we surmise, although as yet the bacteria have not been demonstrated; or it is due to the peculiar zymotic agency or the ptomaines of the other acute infectious diseases, when it occurs as a complication of measles, scarlatina, etc.

Whether alcohol, carbonic oxide, ptomaine poisoning from fish or meat, or the other metallic poisons can produce an acute encephalitis of the cortex, as they are supposed to do in the gray matter of the pons or cord, I doubt; alcohol is the most common direct or indirect cause of Wernicke's form of encephalitis. I should, however, feel inclined to place infection and contagion as the primary if not the only cause of encephalitis of the cortex, although in some of the reported cases of Wernicke's disease, 15 in all according to von Monakow, the cortex and the gray matter of the basilar ganglia have been involved occasionally.

Sexes are equally often affected. If all the cases were published I have no doubt that early childhood up to puberty

would be found to be most frequently affected, principally because the acute infectious diseases are most frequently found at this period of life. Most reported cases are found in adults. General weakness and loss of strength do not seem to be predisposing causes, for, like cerebro-spinal meningitis, primary encephalitis is very frequently seen to attack strong, sturdy, robust individuals, previously healthy. The amount of general exhaustion during the course of an infectious or contagious disease does not seem to bear any relation to the probability of an intercurrent or subsequent encephalitis. Encephalitis of the cortex is most frequently seen during the course of influenza epidemics. During such epidemics primary encephalitis is frequently seen, where the influenza attacks the brain alone; or, to express it more appropriately, the predominating symptoms are on part of the brain, with an absence of respiratory or intestinal symptoms. Encephalitis may occur during an attack of ordinary influenza, or it may follow, either in the course of a few days, or even after weeks or months.

Encephalitis, Lichtenheim's celebrated case, may occur during an epidemic of cerebro-spinal meningitis without any involvement whatever of the meninges. Of the acute infectious diseases I should say that measles is most frequently complicated with encephalitis, next in order typhoid, scarlatina and diphtheria. In these diseases it usually occurs when the disease itself is on the wane, or during convalescence.

Trauma is a frequent cause of acute hemorrhagic encephalitis, and in a twofold way, first in a purely mechanical way. The brain is either bruised by concussion and then a reaction of inflammatory softening occurs, or without any fracture whatever the bruised brain cortex becomes the seat of a circulatory infection; micrococci or spalt-piltze, which are circulating in the blood, are deposited and set up an inflammation in the bruised spot.

Pathological Anatomy—Viewed in its relation to the immediate and remote effects of encephalitis, the pathological anatomy can be considered from two standpoints, the acute stage and the residual stage.

The most frequent form of acute encephalitis in the cortex,

as well as in the gray matter around the canal of Sylvius, is the acute hemorrhagic form according to Fürbringer, who, to my knowledge, at least, described the first cortical cases. The macroscopic appearance of encephalitis is that of circumscribed areas varying in size from a pinhead to a silver dollar; multiple, rarely single, and often symmetrical in distribution, as case No. I will show. These areas are most frequently met in the cortex, but are found also in the white matter of the basilar ganglion. They are bright red in color in the recent cases, but the color varies as the blood pigment changes in color, to a purple-brown, yellowish-brown, or yellow, the area is more succulent than the surrounding tissue, because it is infiltrated with serum, and is slightly raised above the general level of the surrounding cortex and, macroscopically at least, the margins are somewhat sharply defined. Microscopically there is a dilatation of the arterioles and capillaries, and transudation of red and white blood corpuscles into the surrounding tissue, and later on pigment particles, phagocytes and an increase of the spindle cells.

Like unto anterior poliomyelitis the disease is, by concensus of opinion, supposed to be vascular in its origin. What is more interesting to us as neurologists is the fate of the nerve elements, cells and fibers in the area of this acute inflammation. The ganglionic cells are affected, but only secondarily, the protoplasm becomes swollen, more translucent, the nucleus less distinct, afterward the cells become more opaque, and at times the protoplasm is seen to break up, and there seems at times to be a total destruction of cells, at others the cells shrink, become deformed and the nucleus becomes crenated. But this is not a parenchymatous process, but is interstitial in its nature. Aside from the hemorrhagic form Oppenheim has described a yellow or white inflammation in which the macroscopic appearance shows an absence of the engorgement of the arterioles and capillaries, and an absence of the transudation of the red blood corpuscles. In other respects the microscopic anatomy is the same.

The above processes, both the hemorrhagic and non-hemorrhagic may terminte in a complete *restitutio ad integrum*. This is shown not only by the clinical cases in which there has been

a complete recovery from all symptoms, but more satisfactorily by the case observed by Oppenheim. This case was that of a young girl who completely recovered from an attack of encephalitis and died six months later of another disease. The autopsy made by Oppenheim showed a complete absence of all traces of the former encephalitis.

Far more frequently, in the favorable cases which terminate in recovery, some defect of the cortex is found which is the cause of either the incomplete recovery from paresis or of the subsequent development of epilepsy.

The first of these defects is the formation of scar tissue which is seen very frequently. Nerve fibers, especially tangential fibers, are involved in the scar according to a case reported by Buchler. Strümpell contends that the cortical scars seen in children's palsy are due to this form of encephalitis. The second is the formation of cysts. Cortical brain tissue shows a variation in its reaction to inflammation, from the other tissues of the body. In the latter the tendency is to form scars. In the brain the opposite often occurs, cysts being formed, which frequently enlarge by a continuous breaking down of their walls.

In this connection I wish to report a case because, in my mind, it shows what the end state of acute hemorrhagic encephalitis may be, although the early history of the case is lacking and cannot be had.

The patient was brought into the Cincinnati Hospital with the history of having fallen down a flight of steps during an epileptic seizure. He was unconscious and presented hemiplegia of the left side of body, with great rigidity of muscles. The diagnosis of surface hemorrhage was made, the patient was operated upon and obtained relief for a few hours, but subsequently died.

Autopsy—Excessive hyperemia present over surface of both hemispheres. Punctate hemorrhages are seen over surface of both hemispheres, varying in size from a pinhead to a split pea. (Probably of recent origin.)

On the tip of frontal convolution of each side there is a dark spot, about the size of a dime. On section the dark spot is found to be the outer wall of a cyst cavity of about the size of a hazel nut. The wall at one side is thicker and appears laminated. The fluid contents are dark brown in color.

The tempero-sphenoidal lobe on right side shows a similar cyst, but it has ruptured, causing a laceration of the surrounding tissue to size of a hen's egg, and extending to internal capsule, with a large extravasated blood clot. Four other cysts were found on the surface of both hemispheres; all of about the same size. The wall of these cysts are yellowish-brown in color, soft in consistency, in state of disintegration. Fresh hemorrhagic spots are seen in the walls. The surrounding tissue appears soft, granular and infiltrated. The cavities are conical, with apex toward cortex; around the apex there is a laminated structure, dark-brown in color, which microscopically is shown to be an old blood clot.

The pia mater is adherent over some of the cysts. Microscopically there is found in the wall granular debris, leucocytes, a few red blood corpuscles, disintegrated ganglionic cells, giant cells, very little connective tissue.

I have no doubt in my own mind that these hemorrhagic cysts had their origin in acute hemorrhagic encephalitis. This opinion is backed up by Bergmann in a paragraph devoted to the discussion of the treatment of Strümpell's form of porencephalitic defects, in which he speaks of the possibility of these late permanent cysts which occasionally follow encephalitis being the cause of cortical epilepsy and therefore objects of surgical interference. Starr, in "Brain Surgery," publishes a similar case, and Henschen and Dahlgren have collected 17 cases of a similar character which have been operated upon.

That multiple sclerosis may be the late stage of encephalitis is a possibility, but that the clinical disease which we know under this name is the same, is very much doubted, the concensus of opinion being that it is a disease of different origin.

Oppenheim calls attention to the fact that encephalitis is frequently associated with sinus thrombosis and thrombosis of veins, but usually in cases in which clinically there was a well-marked chlorosis.

The clinical history is best illustrated by the following cases:

Case II—Mrs. M., married, mother of four grown children, no miscarriages, has always been well, strong and sturdy. Present trouble began by sudden fainting spell. Dr. C., with whom I saw the case in consultation, found patient in state of extreme collapse, pale, blanched lips, no radial pulse, heart's action very feeble and rapid; no rise of temperature, stimulants were administered and the collapse passed off, and patient, though weak and with a headache, passed several comfortable hours, when a second collapse set in with same characteristics as first. This lasted but half an hour, and on a return of the circulation

patient appeared not only delirious but almost wildly maniacal. She remained in this condition all night and was seen by me on the following morning.

Examination—Sturdy, well-developed woman, 40 years of age, good, healthy nutrition. Mental condition—patient had been very restless all night—constantly attempts to rise in response to idea of going somewhere, going home; answers imaginary voices which she locates as coming from ceiling or the far corner of room, murmurs and talks to herself. Has very few rational intervals; during these she answers questions intelligently, can be aroused to recognize nurse and doctor and her general surroundings. The mental condition, in a few words, was characterized by extreme restlessness, delirium, hallucinations, visual and aural, reactive talk to these hallucinations, and sleeplessness. Pulse 100 to 110, weak, thread-like; temperature 102 to 104; no complaints of pain or headache, no rigidity of muscles, no retraction of head, no opisthotonos. Pupils normal in size, react to light. External muscles normal. Slight paresis of right side of face and tongue. Sensation to touch and pain normal. Upper and lower extremities weak, reflexes normal. No ankle-clonus. Kernig's sign is absent. No Babinski. Passes urine and feces in bed.

The violent delirium occurred at intervals for the first week; during the quiescent period patient was semi-comatose, after third day was no longer rational. The fever continued for the first week; at no time was there rigidity or opisthotonos; after first week there was muttering delirium of a low character, marked weakness of both arms and legs. Patient died on the 14th day. No autopsy.

Case No. III—Henry W. M., *act.* 46, was admitted to "L" neurological of Cincinnati Hospital on April 27, 1898. Discharged May 28. Occupation, carriage maker. Is unable to give any account of himself. No evidence of syphilis. Family state that his illness began ten days ago with headache, dizziness and general malaise. Was treated at first for typhoid. Delirium began a week before his admission.

Present History—Patient was admitted in a state of delirium, muttering in character, with great restlessness, jactilation, fingering of sheets, constant attempt to raise himself with desire to get out of bed, but with an inability to do so on account of general weakness. There is more or less somnolence, with an inability to comprehend questions because his attention cannot be fixed. Seems to have constant aural and visual hallucinations. Temperature normal. Pulse 102, weak, small.

Examination—Mental condition as above. No involvement of pupils. Ophthalmoscopic examination cannot be made. Ex-

amination of muscles of eyes normal. No facial paresis. On account of delirium and inattention, unable to say accurately whether there are any sensory disturbances.

Muscular power diminished all over without any marked localized paresis. Tendon reflexes present. Passes urine and feces involuntarily. After three or four days the delirium became less marked, patient has lucid intervals, and during these it has been found that he has a well marked amnesic aphasia with paraphasia.

May 24—Condition has cleared up entirely as far as sensorium is concerned. Has no delirium, no hallucinations, can understand what is spoken, also reads intelligently. Amnesic aphasia and paraphasia well marked.

May 28—Is able to walk out of hospital, amnesic aphasia and paraphasia still present, and existed to a slighter degree three months later, when patient disappeared from view.

Case IV.—Mrs. F., *act.* 44, married, has 3 grown children, never been seriously sick before. Developed typhoid in March, 1900, ordinary, medium severe case, without any serious complication or malnutrition. During period of febrile decline patient began to complain of headache and vertigo almost at once, not more than a half day elapsing; onset of delirium with hallucinations, especially aural in character, associated with talking in an incoherent manner. The facial expression was that of anxiety and distress, mental in character, an inability to recognize her surroundings. The temperature rose at once to 103, pulse 110. Restlessness, sleeplessness and delirium, together with the high fever, continued for 3 or 4 days. It was noticed that the patient had a difficulty in expressing herself during the state of delirium. After the latter had passed the headache still continued; the patient could be confused very readily by cross-questioning; slightest exertion showed the presence of mental confusion and weakness.

Examination—Mental condition as above, left pupil larger than right, no optic neuritis, both pupils respond to light, no paresis of external muscles of eyes. Slight paresis of lower half of right facial, of right side of tongue and of right arm; right leg not affected. Well-marked amnesic aphasia. Four or five days later the delirium passed away; the amnesic aphasia however continued. The aphasia gradually improved, but never disappeared completely. The difference in pupils also remained.

May, 1901—Patient has had an epileptic seizure. Those who were with her are unable to tell anything more than that the convulsion was general in character and followed by deep coma and sleep. When I arrived an hour later patient remem-

bered nothing of her attack and does not know whether it began in the right arm or face. Patient has been under constant treatment since then and has not had another attack.

The above case is characterized by the mildness of the general symptoms, the rapid disappearance of the delirium and the great local damage done to cortex, viz.: the aphasia, which never disappeared entirely, and the secondary epilepsy.

I have selected the above cases as typical cases, but I would like to call the attention of this Association to another group of cases which we see frequently and of which the following is a type.

Case V.—Mary W., *act.* 15, third child, parents healthy, has never been sick before. Was called in consultation by Dr. B. on account of the development of violent delirium during the third week of a bad case of typhoid. On account of the presence of a very weak heart, and the refusal to take nourishing food, large doses of whiskey were given, together with a 1-40 gr. of strych. sulph., 4 or 5 times a day, the average quantity of whiskey taken during the 4 hours for one week previous to the outbreak of the delirium being 1½ pints. The condition of patient resembled an acute attack of delirium tremens very closely. The delirium was violent, hallucinations massive, no ability to recognize parents or surroundings. Restlessness, tossing, insomnia.

Examination—Girl well developed, strong muscular system, somewhat emaciated, mental condition as described above. Pupils and external muscles of eyes normal. No defect of muscular system or sensation in any part of body. This delirium lasted with more or less severity for a week, but gradually became less and less intense, being displaced by an increased tendency to somnolence and sopor, which state continued for another week without actually passing into stupor or coma. Temperature all this time 102 to 103. Pulse rapid and weak. Respirations rapid, no opisthotonos. Then a gradual return to consciousness with a gradual increase in the amount of food taken, and after 4 or 5 weeks the consciousness became normal and was followed by complete mental and physical recovery, and after five years patient is still strong, vigorous and active.

In another case of this character the stupor lasted for 3 or 4 weeks after the delirium had passed away, and also ended in recovery.

You will see at a glance that the last mentioned cases are not febrile psychoses. I wish merely to call your attention to this class of cases, making only a hypothetical diagnosis, in the absence of localizing signs, placing the seat of the encephalitis

in the frontal lobes, terminating in a complete recovery of the tissue.

The assumption of an encephalitis for the explanation of these cases is strengthened by the experience which we have of the large number of so-calld febrile psychoses which end in incomplete mental recovery, namely, the recovery with mental defect of various grades.

SYMPTOMATOLOGY.

As clearly seen from the foregoing cases, the symptomatology is varied, but the numerous signs and symptoms can easily be grouped under three heads, or rather into three periods, in chronological order, namely: The period of onset and development; (2) the period of focal signs and symptoms; (3) the permanent signs and remote consequences.

The first and second group overlap each other more or less, but are sufficiently distinct to be separated.

The general symptoms may be preceded by indistinct prodromal symptoms for a day or two, such as headache, vertigo, general malaise, but as a rule the onset is sudden, rarely with a chill, usually with a high fever or the exacerbation of a previously existing fever, and there is rapid development of the mental symptoms and the climax is reached in from 24 hours to four or five days or a week. The developed mental symptoms resemble very closely an attack of acute delirium tremens, so much so that Wernicke, who recognizes chronic alcoholism as the most potent factor of encephalitis hæmorrhagica superior, at first was inclined to attribute the mental characteristics to chronic alcoholism rather than to encephalitis. Boedker showed however that this mental state occurred in encephalitis of the cortex without there being any evidence of chronic alcoholism.

Case No. III illustrates perhaps best the general signs of the acute violent form of cortical encephalitis. The cortical form usually affects adults as often as children. The onset in primary encephalitis is usually sudden, with headache and vertigo and a sense of general malaise. A chill, sometimes, though not often, ushers in the rise of temperature, which is at once high, 102 to 104, with rapid, small pulse. Restlessness and a desire to move about and not lie quiet precedes the delir-

ium; collapse, as seen in Case No. II, is not a frequent manifestation. After the development of the delirium, nearly all cases present the same manifestations, the differences in my experience being one of degree and not character. Restlessness, jactilation, sleeplessness, hallucinations both aural and ocular, together with those of general sensation, keep the patient on the alert. Frequently there is a constant muttering of incoherent sentences, but not infrequently there is a response to the aural or ocular hallucinations, calling to supposed friends, answering voices which they hear; frequently maniacal character of movements or speech, constant rambling conversation, smoothing bed sheets, picking at bed clothes, removal of imaginary bugs. In the course of from 2 to 5 days the delirium gradually subsides, the hallucinations and responses to them are not so clearly defined, the speech becomes muttering and incoherent, the acuteness of the fire being smoldered by a blanket of increasing somnolence. The patient frequently becomes lucid at intervals, answers questions promptly, and correctly, but these intervals last only a very short time. During all these days the temperature is high, and the pulse rapid, the breathing is rapid and superficial, there is no, or very little, retraction of the head, though opisthotonos does at times occur, herpes does not develop, and there is no eruption on the body. Sordes of lips and teeth are marked. As a rule there is no change in the pupillary reaction, although in some cases there is an inequality, light reaction usually present, at times absent. Choked disc is always absent in the cortical form, external muscles of the eyes are not involved. Reflexes vary, sometimes normal, sometimes abnormal; some cases are ushered in by general or unilateral convulsions especially in children. In the very violent cases sopor and coma set in and mark most of the localizing symptoms. In the milder cases improvement usually sets in before the somnolence becomes very marked. In the state of coma there is an involuntary discharge of feces, retention and incontinence of urine, and at times bedsores. Death occurs from exhaustion. In those cases which develop during the course of one of the febrile diseases, there is a sudden exacerbation of fever, the pulse becomes rapid, the patient complains of headache and dizziness,

and then almost at once, at least in a few hours, the delirium sets in and the above symptoms may all be developed; or the disease runs a milder course, with delirium, restlessness, insomnia, together with the hallucinations and muttering.

During the course of the delirium, in some cases before it reaches its height, in others after the somnolence has set in, the second group of symptoms is developed. Whether or not these continuous cases of delirium and coma, as illustrated by case No. V. may be looked upon as a manifestation of frontal encephalitis, remains to be seen by future pathological observation. The onset is very sudden, but paralysis is rarely complete, or if it is complete it very rapidly improves during the course of the first few days.

Aphasia, both motor and amnesic, and paraphasia, are seen very frequently as focal symptoms of encephalitis.

In favorable cases the general symptoms clear up, the sensorium becomes free after 2 to 3 weeks, intelligence gradually returns. The focal symptoms, however, remain for some time longer. They may completely disappear in the course of weeks or months, or there may be a permanent defect; and this leads us to the third stage, which is not present in those cases in which the recovery from the inflammation has been complete. The paresis which remains is more or less spastic in character. Contractures, with a retardation in general development, is seen frequently in the cerebral palsies of children.

In adults the paresis may manifest itself as a slight weakness and awkwardness of the arm or leg or entire side.

My experience with the aphasia in both cases, was that the speech showed some defect even a long time afterward.

The most serious of all the sequelæ, however, is the development of epilepsy, as illustrated by Case IV, either Jacksonian, which is usual, or general, which is the exception.

Course, Duration and Termination—The course of acute non-purulent encephalitis of the cortex is that of the ordinary acute infectious disease. Fever is at times absent, but usually a high fever, and all the characteristics of the onset of an acute infectious disease, mark the beginning. The main symptoms of the entire disease show the involvement of the sensorium,

viz., the acute delirium, restlessness, insomnia, hallucinations, followed by progressive somnolence and sopor, and finally coma, show at once the seat of the disease to be in the cortex. The height of the disease is usually reached in 3 to 4 days; then the mental excitement gradually subsides.

Even during the delirium, most frequently during the somnolence, the focal signs make their appearance. After 10 to 12 days the case may end fatally; or improvement takes place, and in a week the sensorium is clear and the focal signs are already waning. Cases are on record in which death has taken place within 24 hours, in other cases in 3 to 4 days; the ordinary duration of the primary acute cortical encephalitis is from 2 to 4 weeks. Those which complicate acute febrile diseases are somewhat shorter, but only apparently because the convalescence of the two diseases is merged together. When death occurs in encephalitis it is due either to the shock of the onset or to ptomaine poisoning in the very acute stages, or is often due to the spread of the disease to vital centers, death occurring most frequently as a result of the involvement directly by inflammatory process of the respiratory or cardiac centers, or of the associating fibers connecting the latter center with the cortex.

In cases which run a favorable course the coma scarcely ever supervenes, even the sopor does not become profound, the case continues for some days, even a week, in a state of mild delirium, with moderate fever and a rapid pulse; when the fever and delirium subside the sensorium becomes clear; the patient shows mental confusion, does not remember clearly the events of the foregoing days; nothing remains aside from this but great exhaustion and the focal signs, which gradually diminish.

Prognosis—In the early stage of our knowledge of encephalitis it was looked upon as almost invariably fatal, but with a broader knowledge of diagnosis, it has been found that the disease very often runs a favorable course, especially where it occurs as a sequela or complication of typhoid or the acute infectious diseases. Pathologically we can have one of three sequelæ, either complete restitution, or the formation of a scar, or cyst. The first is interesting, and has been proven as a possibility by Oppenheim's case. Second and third occur as either the

causes of the permanence of the focal signs or what is equally, if not more important, the causes of subsequent Jacksonian or general epilepsy, and the above data culled from the pathology of the affection is borne out by the clinical data at our disposal. In recovered cases the mental condition is usually good. There may be some of the forms of aphasia, which may lead us to suspect an apparent mental defect. The focal signs usually improve very much. In some cases there is left a slight trace of paresis, and in the two aphasia cases, both of which were amnesic, there was some defect left in speech years afterwards. On the other hand von Jaksch looks upon the prognosis as bad, and doubts the correctness of the diagnosis of the cases which have ended in recovery. In my opinion the prognosis is worse in influenza cases, the result being, as a rule, fatal. My experience is that of Oppenheim, who holds that the prognosis of non-purulent cortical encephalitis is not altogether unfavorable, but at times the disease terminates favorably. Bad prognostic signs are: influenza, rapid onset and short duration of delirium state, with rapid onset of the coma; Cheyne-Stokes respiration, and high temperature. If the delirium is light, the sensorial depression not deep, and the mental symptoms begin to clear up in a few days, the prognosis is good. A very rapid pulse is a bad sign.

The best prognostic indications, therefore, are to be gleaned from the conditions of the sensorium. General conditions must be taken into consideration. During the course of an exhausting disease this general exhaustion may be a bad prognostic sign. But, like epidemic cerebro-spinal meningitis, strong, healthy young individuals may be fatally attacked.

Since von Jaksch is inclined to doubt the accuracy of the diagnosis in the cases with recovery, it behooves us to be very careful in the diagnosis. Oppenheim says that in the vast majority of cases we can only make a problematical diagnosis. We see the importance, therefore, of more exhaustive publication of both clinical and pathological cases.

The diagnosis of the cortical form of encephalitis is much more difficult than that of Wernicke's disease. The differential diagnosis of acute non-purulent cortical encephalitis must

be made from abscess, meningitis, uremic focal lesions, cerebro-spinal meningitis and acute mania, hemorrhage and thrombosis. I speak here of the adult form. In children it must be separated from acute gastro-intestinal disturbance. When the trouble occurs as a primary affection or as a sequela, the diagnostic difficulties are most marked. The most important thing is to rule out cerebro-spinal meningitis. The latter disease is usually characterized by the extreme violence of the headache, and the tendency to spastic rigidity of the extremities; the opisthotonos is very marked, there is a spastic condition of arms and legs, and Kernig's sign is never absent. There is present the delirium and high fever, but the maniacal signs are more marked in encephalitis, the headache is not so severe, opisthotonos usually absent, and retraction of the head, if present, is only indicated slightly; symptoms and signs on part of the cranial nerves in lepto-meningitis involving especially the third nerve, facial and lingual, crossed in character, affecting both sides, whereas these are either absent or only present in one group of muscles, and unilateral. I should lay a great deal of stress on the absence of violent headache and the absence of irritability of eyes and ears, especially during state of delirium, and the absence of uniformly exaggerated reflexes in the encephalitis, specially during the delirium or sopor stage. Cerebro-spinal meningitis may occur sporadically, but as a rule in epidemics, whereas primary encephalitis occurs most frequently during the prevalence of grippe epidemics. It may occur as a sequela of some one of the acute diseases, and then we would rather suspect encephalitis than cerebro-spinal meningitis; monoplegia and aphasia are apt to occur during an attack of cerebro-spinal meningitis, but never stand out so prominently, nor do they occur so early, whereas in encephalitis they occur early and assume the most prominent phase. There is no herpes in encephalitis; we would differentiate from uremic focal lesions first and foremost by a careful urinalysis, but since albumin is sometimes absent in uremic cases, other points would have to be considered. Encephalitis, in the adult at least, where uremia might enter most into consideration, is rarely ushered in by a convulsion, whereas convulsions are the most prominent feature

and occur in rapid succession in uremia. Uremia, in the early stage at least, occurs without fever; encephalitis corticalis rarely without it. Uremic focal lesions occur sometimes before, but usually during the coma period, and usually disappear with the coma, or in a few hours or days after; whereas in encephalitis the focal lesions do not usually make themselves manifest for 2 to 3 days, and persist for a long time—weeks or even months.

It is most difficult to differentiate between encephalitis and acute mania. This cannot be done sometimes for several days. The presence of a continuous fever speaks for encephalitis; moreover encephalitis makes more of an impression of a severe somatic disease, which makes the patient bedfast and exhausts him; whereas the mania case, at least in the beginning, impresses us as more of a mental and less of a physical disease. The onset of the focal signs place the diagnosis of encephalitis beyond question.

Abscess might cause some trouble, especially in those cases where the encephalitis occurs as a complication of one of the acute infectious diseases. An abscess is very rarely sudden in origin, whereas encephalitis usually is; in encephalitis general symptoms, especially those on part of the sensorium or more marked in abscess usually absent, except in the advanced or final stage. The difficulty can only be present in the early stage of either disease, and not in the later stages, and I would place most stress on the condition of the sensorium. Furthermore, in abscess we almost invariably find some local or distinct focus for pus, or at least a history of some injury, usually recent in date.

Hemorrhage and softening are somewhat difficult to rule out, not in the primary affection, for both hemorrhage and cerebral softening usually occur as sudden attacks without prodromata and fever, especially with the typical alterations of the sensorium. Moreover, encephalitis usually occurs in individuals with normal vessels and arteries.

When encephalitis occurs during or as a sequela of one of the acute infectious diseases, it is always accompanied by an exacerbation of the fever; delirium and hemorrhage or softening which might occur during the same diseases usually are not

accompanied by fever or delirium. Oppenheim says that the coma of encephalitis is never deep as that of hemorrhage, and that the pupillary reflex is usually present. Cerebral syphilis is ruled out by the occurrence of a febrile disease.

In the treatment of the cases above, I have used salicylate of sodium in the first few days or weeks; later iodide of potassium and bichloride of mercury.

[2]Magdeburger Naturforscher Versammlung, 1884.
[3]Virchow's Arch., Bd. 38.
[4]Deutsch. med. Woch., 1885, p. 539.
[5]Deutsche med. Woch., 1892, No. 3.
[6]Deutsche med. Woch., 1892, No. 9.
[7]Deutsche med. Woch., 1892, No. 31.

PARAMYOCLONUS MULTIPLEX: REPORT OF A NEW CASE, WITH FURTHER HISTORY OF A CASE REPORTED IN 1896, WHICH HAS SINCE RECOVERED.[1]

By F. W. Langdon, M.D.,
CINCINNATI, O.

The patient, a bright little girl of thirteen, was admitted to my service at the Presbyterian Hospital, February 9, 1902.

Complaint—Muscular spasms, occuring in paroxysms, which began six days previous to admission, and were preceded by pain in the back. This pain followed shortly after a shock from being frightened in play by her brother who was fond of teasing her.

Family History—The father was said to be a dissipated man, who had been separated from his family for some years. The mother, who accompanied the child, was in good health, and not subject to any nervous disorder. She gave a history of seven pregnancies, of which two terminated prematurely, one before and one after the birth of our patient. Three died within a few months of birth.

The brother, the only living child, except the patient, is eighteen, well physically and bright mentally.

Personal History—The patient at birth, appeared strong and healthy and weighed ten pounds, according to the mother's statement. The mother was unable to nurse her at the breast. She had "spinal meningitis" at six months of age, measles at four years, and peritonitis at ten. Had an illness at eleven which was diagnosed St. Vitus' dance, and, which, from the mother's account, somewhat resembled the present attack, but was milder and lasted a few weeks.

Present State—Patient is underdeveloped physically; has defective teeth, suggesting the Hutchinsonian type, but not conforming to it.

Skin clear, no corneal scars.

Temperature, 98.4; pulse, 108, low tension, small volume.

Heart and lungs clear.

Appetite and digestion good, bowels constipated.

Urine passed in bed or with stool, so that it was some days before a specimen was procured for examination. This re-

[1]Read at the annual meeting of the American Neurological Association, June 5, 6 and 7, 1902.

vealed no albumin or other abnormal constituent and was normal in appearance.

The total amount passed, was probably less than normal.

Menstruation has never appeared.

Nervous System—The mental state appears to indicate excessive fatigue. The patient answers questions logically, but very slowly and with great apparent effort. She complains of no pain or discomfort of subjective character, but says "don't hurt me," "don't press so hard," when gently moved about during the examination. This hyperesthesia to ordinary touch seems universal and not confined to localities or zones.

In disposition she is good-tempered and with an unusual degree of patience for a child, not complaining except when touched, and then flinching rather than resisting.

Cranial Nerves—The violence of the muscular spasms, which are brought on by questioning and handling, prevent any detailed tests.

There is no contraction of visual fields to rough tests; no defect of hearing, excepting possibly a hyperesthesia.

Smell and taste are apparently normal.

Trunk and Extremities—Patient lies on her left side, but assumes the dorsal decubitus readily upon request. There seems to be no loss of any voluntary movement but excessive fatigue appears to accompany the slightest exertion.

The grasp is so feeble that it cannot be registered on an ordinary dynamometer.

The muscles of the trunk generally, both dorsal and ventral, and those connecting the trunk and extremities, are the seat of frequently repeated violent clonic contractions . These clonic spasms closely resemble the effect of closure of a galvanic circuit in the muscles.

While these clonic spasms are bilateral, and generally, though not always, synchronous on the two sides, they are not strictly symmetrical in power, being usually much more vigorous on the left side as regards the muscles moving the limbs. At times the difference in activity between the two sides was scarcely noticeable.

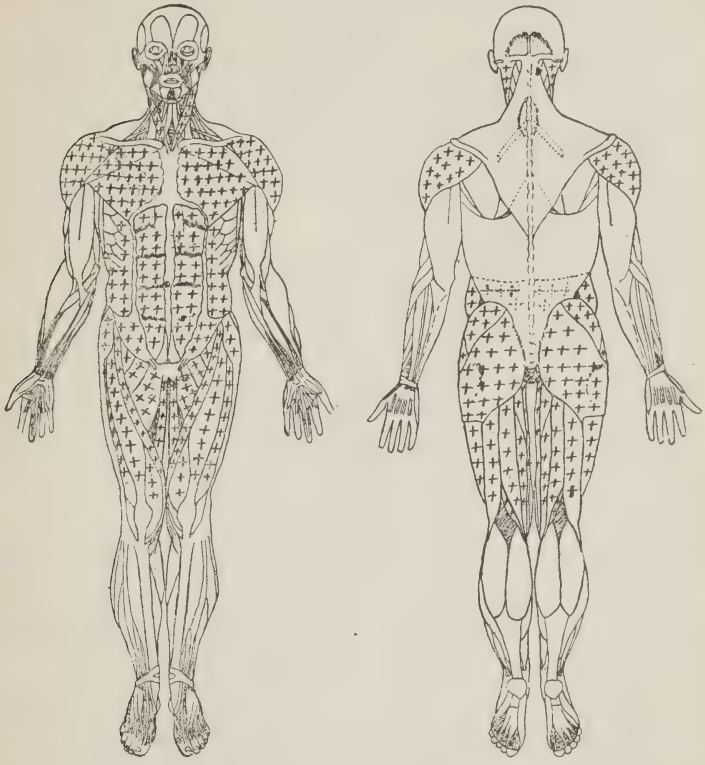
The contractions in the erectores spinae are apparently about equal on the two sides.

A transient opisthotonus is produced by them at times, and on three separate occasions during her first day in the hospital they were so violent as to throw her out of bed on to the floor, which accident was attended with some contusions.

The spasms, which are practically continuous during waking hours, are rendered more violent and more frequent by touching the skin, testing the tendon reflexes, or entrance of another

person into the room; and by attempts of the patient to answer questions.

As regards distribution of the spasms, it is shown fairly well on the accompanying illustration which is reproduced from that made for the first case reported by the writer[2].



Distribution of myoclonic spasm indicated by plus marks.

The chief difference is in the marked involvement of the erectores spinæ in the present case, and their escape in the former one.

The biceps and triceps in the present case are the seat of occasional spasms, while the muscles of the face and neck, the forearms and hands, and the legs and feet seem to escape entirely . The wrists, fingers, ankles and toes remain flaccid, even during the violent paroxysms; their only movements appearing

[2]Cincinnati Lancet-Clinic, February 8, 1896.

to be transmitted from the shoulder and upper arm contractions.

The tongue can be protruded and remain so during the paroxysms; conversation can be carried on, facial movements made at will, without apparent effect, other than the production of fatigue, and some increase in the violence of the contractions of the truncal muscles.

The diaphragm appears affected at times, as evidenced by a short sharp exclamatory cry when any one enters the room unexpectedly, or any noise such as closing a door occurs in an adjoining room.

This is evidently not due to fright, but appears, judging from the absence of accompanying mental disturbance, to be simply a heightened reflex act.

The intercostal muscles do not appear to be the seat of any abnormal movements, and respiration is regular and free.

Contraction of the thigh-muscle groups is about equal in the flexors and extensors, so that the effect is to produce simply a transient rigidity at hips and knees. No movements of rotation, adduction, or abduction of the thighs are observed.

Sensory Symptoms—The patient seldom complains spontaneously, but when questioned says she has pain in the head and back.

Once when the contractions were unusually violent, she asked to be given something to "stop me."

The contractions cease during sleep, but return at any hour of the day or night, if the patient awakes.

Objectively there is no area of anesthesia anywhere, but a condition of hyperesthesia to touch seems generally present. This is especially marked about the head and face.

Reflexes—The visceral reflexes are not impaired. The tendon jerks are highly exaggerated, but do not give the impression of organic disease. Ankle-clonus is absent.

The cutaneous reflexes are likewise much heightened.

The Babinski sign is absent. Testing either the tendon or cutaneous reflexes, brings on the spasms when absent, and increases their activity when present.

The following notes made by the writer at various intervals, show the range of variation in the muscular movements and mental state during her two months' stay in the hospital.

Feb. 8 to 15, contractions chiefly, sometimes entirely, limited to left side. Frequency usually about 55 or 60 per minute, but varying at times between 27 and 98. Occasional intervals of freedom from contractions for from 5 to 10 minutes by the watch.

During this period, hyoscine hydrobromate in 1-200 grain

doses was given every three hours, excepting during sleep. Also potassium iodide in 5 to 10 grain doses.

Previous to her admission to the hospital, chloral and potassium bromide had been given in full doses, with the effect, according to the mother, who had been a professional nurse, of increasing the violence of the spasms.

Feb. 21 to 26. During this period potassium iodide was continued, but Fowler's solution of arsenic in increasing doses up to 12 drops was substituted for the hyoscine.

On Feb. 22, the spasmodic contractions numbered 84 to the minute by two separate counts; were shock-like as usual, bilateral, but rather stronger in the left side; practically continuous except during sleep.

Feb. 26. Spasms irregular in character; first a group of 7 to 10 contractions, rythmical and at the rate of about 60 to the minute; then a change to a group of 7 to 10, much quicker and more violent. Chiefly the glutei and erector spinæ groups are involved.

Mental condition good.

Feb. 27 to March 1. During this period sulphonal in 10 grain doses was given every three hours excepting during sleep.

She received about five doses daily.

Note March 1, 5 P.M. (by writer). Individual shocks less frequent (32 to the minute). At intervals of one to three minutes the shocks occur in groups of 7 or 8, very quickly repeated; then a pause, and a return to the slower rate of 30 or 32 to the minute. Then seven very rapid and violent shocks (sometimes 14 or 15). Then a return to the 32-rate per minute.

The nurse reports that this mode of grouping has been observed for three days.

Patient is delirious, and talkative for the first time since her arrival, and complains of dizziness in head.

Pulse ranging from 72 to 94.

Sulphonal omitted March 1, and no antispasmodic remedies given for three days.

March 3. Pulse 60, low tension, small volume. Spasms in groups of 8 to 15 shocks (rate about 60 to the minute). Between these groups of shocks are intervals of freedom, varying from a half hour to an hour.

Has been singing and elated much of the time; constructs doggerel. Seems good-humored and logical when questioned.

Fluid extract cactus grandiflora in three-drop doses was ordered to improve the circulatory depression.

March 5. Spasms as last noted, but with shorter intervals

of rest (½ minute). Rate 30 to minute with groups of 8 to 15 quicker shocks occasionally.

Mentally somewhat stupid or slow of comprehension.

Asks to be given something to "stop me."

Hyoscine given one dose 1-100 grain and patient slept quietly all night without awakening and without spasms.

Other remedies tried in turn were thyroid desiccated in 3-grain doses; sodium bromide 20-grain doses; conium, Squibb's fluid extract, 3-drop doses; potassium bromide, 10-grain doses; solution bimeconate of morphia, 10 to 15-drop doses.

Each of these drugs was given separately for some days in connection with some tonic medication, but no appreciable improvement could be traced to their use, with the exception of the morphia salt, which lessened the frequency and violence of the individual spasms as well as secured short intervals of entire freedom from them during the waking hours.

The effect on the mental state, however, was bad, as was the effect on appetite, the patient developing an aversion to food of any kind, while before this, all food offered was acceptable and enjoyed.

The spasms continued less violent, 25 to 30 per minute for some days after cessation of the morphine salt, and the mother insisted on removing her from the hospital to her home, where she was placed, by my advice, in the care of Dr. E. M. Keefe, to whose courtesy I am indebted for the following notes:

For the most part the treatment pursued was a tonic one, comprising iron, arsenic, manganese and strychnia in small doses. Occasional doses of hyoscine or bromides were given when the violence of the paroxysms seemed to demand relief but with little apparent effect.

The patient was better and worse by turns very much as when in the hospital. On the whole, however, a gradual improvement took place after removal to her home, so that by May 16 she was free from the muscular shocks for a whole day.

This period corresponded to the one hundred and second day after the onset of the disease.

On the following day, however, the excitement produced by a neighbor in the same house chastising a child, brought back the spasms in full force for 24 hours, after which they have gradually lessened up to date (May 24), when there have been none for 48 hours.

Her general condition is better, as regards color and flesh; she is mentally cheerful and happy, but still so weak as to be unable to sit up.

Further reports of progress and termination are to be expected.

Notes on the Further Progress of a Case of Paramyoclonus Reported in 1896. (Referred to as case one in text.)

Summary.—P. H., male, aged 48, of Irish birth. Onset shortly after a period of business worry, and a mild attack of la grippe.

Distribution of shock-like contractions as shown in accompanying illustration. Frequency of contractions about 70 per minute usually, but varying at times between 50 and 250. Duration of paroxysms from ½ minute to ten minutes.

Number of paroxysms or attacks in a day, varying between 20 at first and two at the latter part of the disease.

Intervals of rest between paroxysms from a few minutes to several hours.

Paroxysms brought on at any time by questioning patient, exposure of surface of body to the air, and by ordinary voluntary movements of the patient.

Remedies used: quinine and arsenic in moderate doses, which appeared at first to lessen the number of paroxysms markedly, but later had no observable effect.

Solution bimeconate of morphia, given in ten-drop doses, at four hour intervals, for a day was followed by almost continuous spasms for 24 hours.

Total time in hospital about two months during which period he went home for eleven days, returning much worse than before.

Hitherto Unpublished Notes of Further Progress of Case 1. *P. H., January,* 1896.

During the eleven days' sojourn at home above noted, the paroxysms of clonic spasms became much more frequent than when in the hospital, and the individual contractions were said to be much more severe. Friends who came with him to the hospital at the time of re-admittance, stated that on one day he was "almost maniacal" in his actions.

A brother-in-law states that he was always a "very impatient" man; hard to control, and likely to over-exert himself at work. During the six weeks following his reädmission, the paroxysms varied in frequency from 8 to 15 for the most part, once going as low as four, and one day (March 20), the spasms were almost continuous night and day, so that separation into definite paroxysms was impossible.

On one day he complained of a "burning sensation in stomach," and on another day, of a "cracking sensation" in the head, but these subjective conditions were not accompanied by any exacerbation in the frequency of paroxysms or the violence of the contractions.

Patient vomited about ½ hour after meals for some days,

and also vomited his medicine, which at this time was sodium phosphate. On one occasion he had a moderately severe pain in the abdomen, for which solution of morphia bimeconate was given in small doses. This, while apparently benefiting the abdominal pain, was followed by a pain in the "back of the head," and by considerable increase in the frequency and severity of the spasms. He left the hospital little if any improved as regards the spasmodic contractions and disappeared from view for nearly two years.

On Jan. 25, 1898, he called at my office apparently in perfect health, having gained 25 lbs. in weight, and been free from spasms of any kind for eleven months.

He stated that after leaving the hospital, March 1896, he continued treatment at various places for about a year, but with no benefit to the spasmodic contractions.

He ascribes his recovery to his return to hard laboring work (ditch digging), which he resumed a year ago (1897), after discarding all medical treatment.

Since this note, I have seen him in May 1900, and again in January 1902, and he has had no relapse nor ill health of any kind during these intervals.

NEW YORK NEUROLOGICAL SOCIETY.

April 8, 1902.

The President, Dr. Joseph Collins, in the chair.

A Case of Asthenic Bulbar Paralysis.—Dr. George W. Jacoby presented a young woman with asthenic bulbar paralysis. He said that this was only the second case that he had seen. The other case was that of a young man who now had an intermission in the disease that had lasted for three years. From the histories of other reported cases it was not probable that the man had permanently recovered. The patient presented was a girl of twenty years, who had been perfectly well until last May, when, while reading aloud, her voice was noticed to falter. Within a few weeks this became quite noticeable even on speaking. About this time there was some double vision. Later on there were nasal speech and regurgitation of fluids through the nose. Only within the last two months had she developed a slight weakness of the right hand. Dr. Jacoby called attention to the broadness of the mouth, the thickness of the lips, the almost expressionless appearance of the face, and the difficulty of pouting the lips as in the act of whistling. There is inability to completely close the eyes, and the reflexes are exhaustible.

An Experimental Study of the Reflexes in Total Transverse Lesions of the Spinal Cord.—Dr. William Aldren Turner, of London, presented a paper on this subject (see page 321).

Dr. Philip C. Knapp, of Boston, said that comparatively few of the writers upon the reflexes had dwelt upon one point, *i.e,* the extreme difficulty of proving anatomically the fact that the cord is totally destroyed. In one of his own cases there was clinical proof of conduction through the cord, and sections were made with very great care in order to determine the condition of the softened portion of the cord. It was found extremely difficult to make out the presence of normal nerve fibers in the softened part although undoubtedly there must have been some. This might explain some of the published cases which appeared to contradict the rule. A case had come under his observation in which on removing the fractured laminæ the cord was found absolutely divided and the ends free. In this case a year after the accident there was flaccid paraplegia with loss of the knee-jerk and retained plantar reflex. This occurred before attention had been called to the Babinski reflex. Bastian's theory of the influence of the cerebellum upon the reflex seemed to be weakened by the fact that so rarely was there any change in the reflex in cases of cerebellar disease. The explanation of loss of the knee-jerk from complete section of the cord seemed to him far from satisfactory. It was known that if the entrance of the sensory fibers in the posterior horns and columns in the third and fourth lumbar segments were destroyed, and the corresponding cells and anterior roots innervating the quadriceps were destroyed, the reflex was lost, but even assuming that the nerve impulse must pass into the cord to the level of the cervical enlargement, or possibly higher, it was difficult to understand this by the experiments brought forward by Dr. Turner. If there were a reflex arc or a mechanism for the neuromuscular tonus, why should it be destroyed by a lesion high up while a lesion below, which

must cut off all communication, allows of the preservation of the reflex?

Dr. Charles K. Mills, of Philadelphia, said that he did not feel that he could contribute anything new to this interesting subject, but he would mention the fact that he had seen quite a number of cases, with autopsies, which tended to confirm the views expressed by Dr. Turner. He had had a number of autopsies on cases of incomplete section of the spinal cord in various regions, in which the conditions were those already described by Dr. Turner. He could recall several traumatic cases in which high lesions with complete loss of the knee-jerk had been present. His mind was far from being clear as yet with regard to the mechanism allowing of the retention of the knee-jerk under certain conditions and its loss under others. The cerebro-cerebellar theory had always seemed to him the most nearly satisfactory. He had had a comparatively large number of cases of cerebellar lesion, and these had shown interference with the knee-jerk, but the conditions had not been uniform. He had seen loss of the knee-jerk in cases afterwards demonstrated to be examples of cerebellar disease. He had seen increased knee-jerk and even crossed knee-jerk in connection with cerebellar disease, and he was satisfied that disease of the cerebellum plays a part in interfering with the knee-jerk as with other reflex phenomena. The fact that lesions at the cervico-dorsal junction when complete were apt to cause loss of the knee-jerk, was of great interest. The existence of a ciliospinal center in a particular region of the cord might be suggestive with regard to the effect upon the knee-jerk and the lower reflexes in these particular cases.

Dr. Joseph Fraenkel thought, with Dr. Mills, that the more this problem was studied the less seemed to be really known about it which would be of value at the bedside. The presence of a ciliospinal center high up in the cord might help us in the explanation of these phenomena. A number of heterogeneous phenomena were considered in connection with the reflexes. The plantar reflex, which seemed to be the only permanent skin reflex, was representative of the skin reflexes just as much as the knee-jerk was characteristic of tendon-reflexes. This knee-phenomenon was one of the expressions of the muscular qualities. A muscle possesses what is called irritability, and quite a complicated neural apparatus keeps up what is called muscular tonus. Whatever interfered with this influenced the tendon reflexes. In conjunction with the President of this Society he had carried on for four years a clinical study of the reflexes, measuring at the same time the tonicity of the muscles. This study embraced about 600 cases, and included certain interesting pathological findings. They had become convinced that the knee-jerk was absolutely dependent upon the muscle tonus. If the muscle tonus were increased through pain in the joint there would be an increase of the knee-jerk. When the lesion of the spinal cord had been slowly progressive there would be nutritive shortening of the muscle, but the muscle tonus not being destroyed the reflex would still persist. The tendon reflexes should really be placed in the same category with the visceral reflexes. The considerable discrepancy between the findings at the bedside and in the laboratory had been explained by a recent publication of Strümpell, in which it was stated that the mechanism of the reflexes in the lower animals and in man was different.

Dr. M. G. Schlapp referred to a reported case in which, on histological examination, the cord was found to be absolutely severed in the dorsal region. This case overthrew the Bastian theory. He thought the reflexes should be considered in their abnormal conditions. If a poison were introduced into the body it would exert a selective action on the neurones of the central nervous system. In cases of ergotism, lead poisoning, typhoid fever and brain tumor, degenerations were found in the

posterior columns and in the roots entering these columns. If these fibers were traced, they would not be found to pass into Goll's columns, and consequently they were short fibers. In many cases of brain tumor in which the tendon reflexes were missing there would be no sensory disturbance. These fibers were probably the sensory half of the reflex circuit. The loss of reflexes, he thought, could be explained by degeneration of the short neurones in the posterior roots having to do with the tendon reflexes. He had found this lesion of the cord in cases of transverse lesion of the spinal cord, and also in cases of brain tumor, and in a case of alcoholism. These were facts, and consequently demanded more careful attention than mere theories.

Dr. Joseph Collins said that his own views had been quite well expressed by Dr. Mills and Dr. Fraenkel.

Dr. Turner, in closing, said that until he had collected the results of his experiments in tabular form he had had no idea that the condition of the reflexes was so extremely variable. On referring to the literature he had found that this had been noted by several observers quite a number of years ago. He was in perfect accord with the position taken by Drs. Collins and Fraenkel. The condition of the knee-jerks was entirely due to the state of neuromuscular tonus, for it was just this which was affected by transection. The superficial anal reflex was a pure reflex, and was found usually in monkeys and commonly in man. Unfortunately, the plantar reflex could not be elicited in monkeys. The effects on the reflexes observed in cases of disease of the cerebellum and in experimental lesion of the cerebellum were very different. He thought we should ascribe these variable phenomena not so much to the existence of a tumor in the cerebellum as to interference with intracranial pressure from the presence of a morbid growth under the tentorium cerebelli.

PHILADELPHIA NEUROLOGICAL SOCIETY.

April 22, 1902.

The Vice-president, Dr. F. X. Dercum, in the chair.

A Case of Friedreich's Ataxia was exhibited by Dr. William G. Spiller.

A Note on Some Psychoses of Early Puberty, with Report of a Case in a Boy Twelve Years Old.—This paper was read by Dr. A. Gordon, who remarked that in cases where children were described as unusually bright, he looked upon them with suspicion as regards the development of psychoses. Similar observations have been made by others.

He also emphasized the great instability of the mental state in these patients. In his case subsequent to the excitement caused by a fire the symptoms immediately returned. This is analogous to what is seen in organic disease of other organs of the body.

Dr. William Pickett regarded Dr. Gordon's case as an interesting one from many points of view. Dr. Gordon had referred to a similar mental state in one or both parents. A similar heredity was pointed out in these cases by Morel. Looking at the condition from that point of view Dr. Pickett could understand why Dr. Gordon should follow Magnan and others in classifying this young man as an example of the psychic stigmata of degeneration. He had seen a good many of these cases complicating other forms of insanity or incidental in other forms of insanity. As the speaker had pointed out in his paper on dementia præcox, there is an objection to calling these manifestations psychic stigmata, as they come and go according to the physical condition, as is exemplified in the present case. He believed that these cases recover, particularly at this period of life. That fact makes it more rational to classify these cases as neurasthenics. We usually find some of the familiar symptoms of neurasthenia in these cases, and so neuropathic neurasthenia seems the most expressive term for this group.

Another question of importance in connection with these cases, is how much use can we make of these stigmata for purposes of prognosis. Does the early presence of obsessions render it more probable that a young man of unstable makeup will later become the victim of some form of adolescent insanity? Dr. Gordon predicts that his patient will become a case of dementia præcox. Kraepelin asserts that the percentage of permanent dementias following mental disturbance at this period is larger than ordinarily supposed, from the fact that the dementia is often slight and manifests itself largely in the moral sphere by a change of character.

It was for such cases, which we might call moral dementia, that Kahlbaum proposed the name heboidophrenia.

Dr. F. Savary Pearce said that the case reminded him of one which had come under his observation a few days before at the Medico-Chirurgical Hospital. The patient was an Italian boy about twelve years of age. There was a history of degeneracy in the family. There is no doubt that this boy is the subject of moral insanity. He had been in the habit of knocking other boys about, and had himself been knocked about by the father of one of the boys whom he had abused. Following this he had pseudo-epileptic attacks. The father assumed that the injury was the cause of the so-called fits and the mental degeneration, and proposed

to bring suit. The speaker had told the parent that the injury was not the cause of the trouble,—that the boy was morally defective and should be in a hospital.

He had seen another case in a young girl with a history of insanity on the mother's side, and he regarded this as a case of dementia præcox. She had had three attacks of dementia. The present attack is peculiar inasmuch as she will not speak of persons except as numbers. The girl has been overworked at school, particularly in mathematics.

Often in these degenerate subjects the delusions and hallucinations are associated with particular lines of overwork. His experience was that these patients are not usually intelligent, although the family will often say that they are, and perhaps they are precocious as compared with other children in the family. He did not believe that precocious or bright children in normal families are more liable than others in the family to become insane. He referred to a young woman of his acquaintance who as a child was very precocious. She had grown to perfect womanhood and was very bright.

Dr. Alfred Gordon stated that what he had said about his patient being unusually bright did not rest entirely on the statements of the family. Their evidence had been confirmed by the boy's teachers. His experience had been that cases of unusual brightness should be looked on with suspicion.

Dr. Charles W. Burr agreed with Dr. Gordon that preternaturally bright children should be looked on with suspicion. Any one who has had much to do with criminal children knows that they are plausible and that they can lie after a fashion that older persons cannot, and that shows their bright character.

An Unusual Case of Myasthenia.—This paper was read by Dr. David Riesman.

Dr. D. J. McCarthy said that in a case reported by Dr. Burr and himself there was distinct smallness of the medulla oblongata and the spinal cord. It was the smallest pons by one-third that he had seen. In another case not reported there was a distinct small abscess in a persistent thymus gland. In a case reported lately there was a persistent thymus gland. In a recent autopsy reported there was a persistent thymus with tumor formation and round-cell infiltration into the muscular tissue which proved to be metastatic from the thymus gland. In one case the myasthenia followed pregnancy. The toxic theory seems to be the only plausible one to account for these cases, but there is as yet no tangible evidence to support it.

Dr. A. C. Croftan favored the view that Dr. Riesman's case was one of Addison's disease without pigmentation. He had seen a similar case in Vienna in 1896. The patient had an attack of myasthenia from which he recovered. Some six months later he died from general tuberculosis. One of the adrenal glands was destroyed by the tuberculous process, and the other was greatly hypertrophied. The myasthenia may have resulted from the atrophy of the gland and was later relieved by the compensatory hypertrophy of the other gland.

The President suggested that Dr. Riesman, in his closing remarks, should indicate the points of differential diagnosis between his case and one of Landry's palsy.

Dr. Riesman said that he looked upon his case as one of grave myasthenia due to functional insufficiency of the adrenals. He was quite convinced that the man had no tuberculosis anywhere in his body.

With regard to the distinction from Landry's palsy, the case reported by him was not, he thought, one of paralysis in the true sense; the man could lift his limbs and could perform all the finer movements with

his hands, only, however, with great difficulty, on account of great weakness. If given time he could accomplish the movements he wished to make. There was no foot-drop; no loss of knee-jerk. There were no phenomena indicating involvement of the respiratory muscles, nor did the symptoms show the characteristic ascending tendency. The physicians in attendance had considered the case one of neurasthenia; and, for practical purposes, this was a good diagnosis. There were no sensory symptoms, and the patient recovered in two months.

A Case of Tumor of the Pons, Medulla Oblongata and Upper Part of the Spinal Cord with Necropsy was reported by Dr. W. G. Spiller.

Dr. Alfred Gordon referred to a case reported by Oppenheim. The patient had been under observation four years, complaining of pain in the back. It had been diagnosed as rheumatism. Oppenheim insisted that there was a tumor of the spinal cord and this diagnosis proved to be correct.

A Case of Attempted Self-Castration was reported by Dr. A. R. Moulton.

Dr. James Hendrie Lloyd said that this case was of great interest and like one which he had seen at the Philadelphia Hospital years ago when he was examiner of the insane. The man, under the delusion that he was wasting away, took out one testicle. He was taken to a hospital and recovered. In the course of a few months he removed the other testicle. After recovery from the injury he was sent to the Philadelphia Hospital as an insane patient. He was then exceedingly insane, showing that the removal of the testicles in response to an insane delusion was not curative. So much was he impressed with the idea that he was losing his virility that one day, securing a knife, he made a deep gash in his perineum. He did this because he believed that there was still some secreting tissue left, in spite of the fact that both testicles were gone. He was again treated and again recovered from the wound. These cases should be forced upon the attention of our gynecological friends. The gynecologist who takes out the ovaries of an insane woman with the idea of curing her insanity does just what this man did on himself. The one is just as mad and unphilosophical an idea as the other—just as insane.

There is another important point in this connection. There has come to be a cult of people who are recommending the "open-door treatment" of insanity. Throw open the doors, they cry, throw away your locks and keys, and let your insane people go free. They carry to an extreme the views of Tuke and Pinel, who struck off the shackles of the insane. These cases of self-mutilation illustrate how absolutely improper such a procedure would be.

Dr. F. Savary Pearce mentioned a case which had been described to him by a physician of a man perfectly sane who could not afford the fee for removal of the testicle for disease, and who had operated on himself successfully.

Drs. C. K. Mills and W. McConnell exhibited a patient showing a new group of brachial and pectoral reflexes. Tapping over the inner aspect of the shoulder and also at the points where the second and third ribs join the sternum caused certain reflex movements in the arm, forearm and fingers They had found the same reflex phenomena in normal individuals, and the same exaggerated or impaired in a number of patients suffering from organic nervous disease. The increase, diminution or loss of these reflexes occurred under the same conditions of disease as caused similar changes in the von Bechterew scapulo-humeral reflex.

Thrombosis of the Midcerebral Artery, Causing Aphasia and Hemiplegia, with Remarks on Central Skiagraphy.—This paper was read by

Dr. C. W. Burr and Dr. G. E. Pfahler. In connection with the paper Dr. Pfahler exhibited skiagraphs from the brain. He had up to this time made fifty-one skiagraphs of brains and believed that these examinations would be of service in the diagnosis of diseases of the brain.

Thrombosis of the Posterior Limb of the Midcerebral Artery.—This specimen was exhibited for Dr. C. K. Mills.

A Case of Ataxia with Recurrent Clonic Spasm in the Anterior Tibial Muscles was reported by Dr. J. H. W. Rhein.

A Specimen from a Case of Hydrocephalus was exhibited by Dr. F. Savary Pearce. The patient, a colored child aged two and a half years, had been ill about nine months. There was no tuberculous history in the family. There was no evidence of tuberculosis in the brain. The lateral ventricles contained about a pint and a half of clear serum. Every bone in the cranium was disarticulated. The child died of inanition, following a series of slight convulsive seizures, tetanic in nature.

Periscope.

Deutsche Zeitschrift für Nervenheilkunde.

(1902. Band 21, Heft 3-4.)

7. A Case of Asthenic Bulbar Paralysis with the Results of Autopsy. Liefmann.
8. The Psychical Disturbances of Tumors and Injuries of the Frontal Lobes. B. Müller.
9. The Centrifugal Impulse in the Sensory Terminal Neurones. Kohnstamm.
10. Operative Interference in Choreic Epilepsy. Von Bechterew.
11. The Results of Almost Complete Strumectomy. Lundborg.
12. Bulbar Paralysis and Sarcomatosis. Hensen.
13. Tuberculous Meningomyelitis. Hensen.
14. The Localization of Reflex Pupillary Immobility. Wolff.
15. Partial Myotony with Muscular Wasting. Schott.
16. A Contribution to the Question of Infantile Tabes. Idelsohn.
17. Remarks upon the Clinical Observation of the Skin and Tendon Reflexes of the Lower Portion of the Body. Schoenborn.
18. The Changes in the Spinal Cord as a Result of Intra-cerebral Pressure. Finkelburg.
19. Brief Communications. (1) Contribution to the Knowledge of the Reflexes. Bickel. (2) The Peculiar Associated Movements of the Paretic Palpebral Elevators and Sphincters. Higier.
20. Book Reviews.

7. *Asthenic Bulbar Paralysis.*—Liefmann reports the case of a girl nineteen years of age, without neuropathic heredity, who, five years after an attack of diphtheria, during which she was severely ill, developed paresis of some of the muscles of the face, particularly the left eye-lid. A year later there was bilateral paralysis of the muscles of the face, and finally paralytic manifestations in all the motor cranial nerves, giving rise to external ophthalmoplegia, facial diplegia, and disturbances of speech, swallowing and mastication. At the same time the patient had fatigue of all the muscles of the body, atrophy of the right half of the tongue, and some quantitative alteration in the electrical reaction of the muscles supplied by the facial nerve, that is to say, the patient presented the characteristic picture of asthenic bulbar paralysis. She died rather suddenly, and macroscopic changes were not found in the central nervous system. The histological examination showed practically no changes in the cells of the central nervous system, with the exception of some peculiar homogeneous masses, resembling colloid, which were found in the perivascular spaces of the capillaries, and scattered through the nervous system. The muscle of the tongue showed a considerable infiltration with fat. Liefmann discusses the nature of asthenic bulbar paralysis and quotes a number of cases from the literature. He believes that the disease may be associated with atrophy of the muscles, and that this atrophy may be due to a peculiar noxious substance that interferes with the function of the central nervous system. This noxious substance is probably toxic in character.

8. *Psychical Troubles and Frontal Lobe Tumors.*—Müller discusses particularly the case reported by Welt, which is supposed to indicate that disturbances of intelligence are apt to follow lesions in the frontal lobe. He believes that in all such cases a diffuse disease of the cortex of the brain should be absolutely excluded, and that the patient should be proven to be not of a neurotic character before the injury or tumor developed. In a case reported by Welt there is every reason to believe the patient was hereditarily defective, and in many other cases in which the proof is entirely inadequate that the frontal lobes are particularly involved this explanation may be the true one. In regard to the frequency with which a tendency to make puns occurs in cases of disease of the frontal lobes, Müller, after admitting it, attempts to explain it by various hypotheses. (1) That the tumor may produce a general dementia; (2) that it may be the result of epilepsy; (3) that certain chemical substances may be generated by the tumor, or administered as remedies; (4) it may stimulate and appear excessive on account of the contrast with the general apathy and indifference of the patient. He concludes, therefore, that the symptom is only a general symptom of tumors of the brain, is caused by the progressive dementia produced by the tumors, and is more common in tumors of the frontal lobes because their course is relatively less acute. The difficulty in the discussion of this subject lies in the fact that a very large amount of material has been used for statistical purposes, and that much of this material is not sufficiently conclusive. The majority of brain tumors in this region are gliomas, or gliosarcomas, that is to say, they are probably congenital in nature. These tumors may be associated with psychical symptoms in three ways: either by interfering with the general nutrition of the brain, or the psychical condition and the tumors may arise from the same cause, or the psychoses may preëxist, and, by predisposing to injury, act as a remote cause of tumor formation. In conclusion Müller urges that in future all cases of brain tumor be carefully studied by the psychiatrist in order that our information regarding the relation of these tumors to mental defects may be more accurate than it is at present.

9. *Centrifugal Impulse in Sensory Neurones.*—Kohnstamm believes that the centrifugal neurones may be irritated by the central nervous system. The arguments in favor of this may be summarized as follows. (1) The phenomena of the reflexes which may extend from one posterior root to another; (2) trophic disturbances which occur in organic disease and in experiments upon animals; (3) the pathology of herpes zoster; (4) the fact that groups of nerve fibers exist in the midst of sensory neurones, carrying impulses toward the periphery; (5) the changes in the spinal ganglion cells after section of their peripheral fibers, and (6) the fact that the ganglion cells apparently may conduct impulses in two directions. Each of these points is discussed in a separate paragraph, and Kohnstamm finally concludes that the hypothesis is sufficiently clear.

10. *Choreic Epilepsy.*—Von Bechterew reports the result of an operation upon a case of choreic epilepsy, a condition that he has previously described. The patient showed cramp-like twitchings in various parts of the body, occurring before the epileptic attacks, the latter frequently giving rise to serious injury. At the operation the skull and dura were permanently removed from the Rolandic region on one side. The irritability of the cortex was carefully tested, and then small portions of the gray substance of the central convolution removed at three places. There were no signs of paresis after the operation, and the patient was so remarkably improved that he was ready to submit himself to an oper-

ation upon the opposite side. A similar operation was therefore performed, but unfortunately, as a result of erysipelatous infection of the wound, the patient died.

11. *Strumectomy.*—Lundborg, in discussing the results of strumectomies, involving four-fifths or more of the thyroid gland, states that there may be 6 terminations: (1) Acute tetany, followed by death; (2) acute tetany, followed by recovery; (3) chronic tetany; (4) chronic tetany and myxedema; (5) myxedema; and (6) entire recovery. He reports the case of a woman twenty-four years of age, with neuropathic heredity, who, in childhood, had developed goiter of both sides of the neck. She also had (even as early as the age of eleven years) tremor and tachycardia. At the age of 15 a portion of the left lobe was removed, and at the age of 17 the right lobe was also removed. Almost immediately after the second operation the patient developed acute tetany, which gradually passed ino a tetanoid condition and then improved. A certain amount of renewal of the tremor occurred. The attacks of tetany, however, continued to occur especially at the menstrual periods; they were preceded by an uncomfortable feeling in the arm and then by clonic spasm. These clonic movements involved all the extremities and sometimes even the whole body. The patient was given thyroid tablets and gradually improved, although she did not entirely recover.

12. *Bulbar Paralysis and Sarcomatosis.*—Hensen reports the case of a girl, nineteen years of age, who had an attack of some acute condition, accompanied by fever and vomiting. Three weeks later, she felt as if the lips were thickened, and a week after this there was paralysis of the left side of the face associated with paralysis of the abducens nerve, atrophy of the tongue, a nasal and hoarse voice, difficulty in swallowing, and slight paresis of the right side of the face. The liver and spleen were enlarged, but otherwise no pathological condition was discovered, with the exception of the total cessation of menstruation. The patient slightly improved, but developed necrosis of the right tonsil, associated with slight fever. She then became anemic, rapidly grew weaker, and died. At the autopsy there was found sarcomata of both ovaries with metastasis to the spinal column, the liver, kidneys, uterus and vagina. The central nervous system was peculiarly firm in consistency; there were some grayish white lines in the white substance of the brain; slight hydrocephalus and a doubtful tumor-like formation upon the outer side of the dura. The microscopical examination showed peculiar changes in the central nervous system, particularly in the medulla, which led to the suspicion that they might be beginning metastases. Small focal lesions and extensive secondary degeneration of the nerve fibers could be traced. As, however, these focal lesions appeared to be more recent than the symptoms, it is possible that some other cause was acting at the same time.

13. *Tuberculous Meningomyelitis.*—Hensen reports the case of a woman forty years of age, who had chill, headache, severe pains in the back, difficulty in urination, and, when brought to the hospital, she showed slight paresis of the right abducens, paralysis of the bladder, hyperesthesia of the legs, and slight increase in the patellar reflexes. The patient improved slightly, and then developed numbness in the lower limbs, with paralysis in both. She had also girdle pain and gradual ascent of the anesthesia along the body. Reactions of degeneration occurred in some of the muscles. The patient finally died, and at the autopsy tuberculous basilar meningitis was found. A tuberculous myelitis was found about the middle of the dorsal region, which produced almost complete destruction of the cord. The case is rare, and

apparently commenced as a meningitis of the spinal cord, followed by the myelitic changes.

[A note by the editor states that the author of these two articles died of tumor of the brain before they appeared.]

14. *Localization of Reflex Pupillary Immobility.*—Wolff reports the case of a man forty-five years of age who, a few months before admission, had had pain in the back between the shoulder blades; this gradually extended to the neck and head. The patient developed diplopia and twitching in both left extremities. Memory was much impaired; he gradually developed paresis of the left leg, and then recovered almost entirely after employing potassium iodide. He then became worse, was completely irresponsible; the pupils were of the Argyll-Robertson type, and there was paresis of the whole left side of the body. The patient gradually grew weaker, developed cough with expectoration, and finally died. At the autopsy a large tumor was found in the right cerebral hemisphere, extending almost to the wall of the third ventricle, and involving the corpus striatum and the anterior portion of the optic thalamus. The central portion of the brain was sectioned by the Weigert method, and two other gummas were found by this method, one of the ventral surface of the brain at the junction of the pons and medulla, and the other in the upper portion of the cervical cord, extending from the second to the fourth segments. Wolff discusses the difficulties in locating the lesion that gives rise to the Argyll-Robertson pupils. Theoretical explanations are unsatisfactory. In his cases Westphal's nucleus was not involved, and there seems, perhaps, more reason to suppose that the cervical lesion had more to do with it than any of the others. He admits that at present we know nothing at all about it.

15. *Partial Myotony.*—Schott discusses the case of a boy eighteen years of age, who had myotonia, commencing in the legs and then extending to the hands. The symptoms were more pronounced in cold than in warm weather; otherwise the patient was normal. Electrical reactions showed no particular abnormality, excepting in some of the muscles of the fingers, in which there was slight prolongation of the contraction, a marked reduction of faradic irritability, and an inversion of the galvanic formula. The disease does not appear to have occurred in any other member of the family.

16. *Infantile Tabes.*—Idelsohn reports a case of a girl six years of age, whose father and mother suffered from syphilis, who had Argyll-Robertson pupils; the tendon reflexes were lost; station and gait were normal; there was hypalgesia in the legs, and the child slept more than usually. There seems little reason to suppose that the case is anything but tabes dorsalis. Altogether Idelsohn has been able to collect six other cases occurring in young children. In all there was leutic infection of the parents and the characteristic symptoms.

17. *Skin and Tendon Reflexes.*—Schoenborn, after a discussion of the individual reflexes and the methods in which they are elicited, in the course of which he touches upon several interesting topics, as for example, whether the knee-jerks are ever present in tabes,, mentioning an interesting case in which the symptoms were fairly typical, but in which very marked improvement subsequently occurred, and also the infrequency with which he has been able to obtain patellar clonus, then proceeds to the discussion of the skin reflexes, of which he mentions a considerable number. Among the most important of these is the Babinski toe phenomenon. He then gives in tabular form the results of his investigations in one hundred persons, who were apparently not suffering from nervous disease. These are as follows:

	Present	Doubtful	Absent
Patellar reflexes	100	—	—
Achilles tendon reflexes	97	2	1
Adductor reflexes	38	8	54
Tibialis posticus reflex	22	6	72
Abdominal reflex, upper	98	1	1
Abdominal reflex, middle	99	1	—
Abdominal reflex, lower	98	1	1
Cremaster reflex	98	1	1
Anal reflex	80	7	13
Scrotal reflex	92	3	5
Plantar reflex	98	1	1

18. *Intracerebral Pressure and Cord Changes.*—Finkelnburg reports three cases of cerebral tumors occurring in children, all of which were in the cerebellum. The common characteristics of these cases were extreme intracranial pressure, with papillitis, loss of knee reflexes in two cases, early death, and at autopsies characteristic changes in the posterior and anterior roots. The ganglion cells in the anterior cornua, however, showed no changes, and there was no reason to suppose than any toxin was acting on the spinal cord because all the changes could readily be explained by the supposition of increased intraspinal pressure. Finkelnburg has now attempted to discover by experiment whether the intraspinal pressure is sufficient to account for all the changes found.

19. *Reflexes.*—A. Bickel reports an interesting experiment performed upon a dog. The posterior roots coming from the posterior extremities were cut, and it was found that there was total anesthesia in the hind legs, with loss of the tendon reflexes. After this condition had persisted for five months, transverse section was made in the dorsal spine, when it was found that irritation of the posterior roots caused movements in the extremities and in the tail. As the microscopical examination showed that no regeneration in the posterior roots had occurred, Bickel believes that the results are best explained by assuming that fibers from the hind legs enter the spinal cord at a higher level, and that the irritation of these produced in the hyperexcitable motor tracts the movements of the extremities.

(b) *Paretic Palpebral Elevators.*—Higier describes some peculiar movements in the eyelids occurring in a man 32 years of age. Under normal conditions the right eye was covered, and the left was abnormally wide. When he began to chew the right eye opened and the left closed. The patient had paresis of the right levator palpebræ, and in the left orbicularis oculi, otherwise his muscles were normal, excepting that the ptosis had existed from birth and the lagopthalmus only for 9 months. As the right eye could not be raised voluntarily, and was only elevated when the mouth was opened, it was necessary to make a careful study of the muscles, when it was found that the pterygoids and the muscles of the hyoid bones were the ones whose contractions were of special influence. The closure of the left eye is probably to be regarded as an associated movement. J. Sailer (Philadelphia).

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1. Examination of Paraphasia. G. Saint Paul.
2. Considerations of Late Epilepsy and Senile Epilepsy. Paul Masoin.
3. Suicide and Insanity. Viallon.
4. The Language of Idiots. L. Maupate.

5. Protection of the Fortune of the Mentally Deranged in Asylums. What it is, and what it should be. SAMUEL GARNIER.

1. *Examination of Paraphasics.*—The author discusses at great length the different types of aphasia, and devotes special attention to paraphasia, which syndrome he elucidates by a series of diagrams. He proposes a lengthy, complicated, and valuable series of tests designed to further dissociate many heretofore confused mental phenomena. Of the different forms of paraphasia, he distinguishes: (a) *Paraphemia*, the principal symptoms of which are dysphemia, dyslexia, and motor dysechophemia. In this form the faculty of oral reading may or may not be conserved; the faculty of mental reading may be similarly affected. Faculty of repetition is perfect. The influence of previous occupations should be taken into account. Other symptoms referable to the author's diagram, are given.

(b) *Paragraphia*, of which the main symptoms are dysgraphia, dyscopia, and motor dysechographia. The same series of tests for paraphemia are here applicable.

(c) *Associated Paraphemia and Paragraphia.*

(d) *Verbal Paracecité* (Word Blindness). The principal symptoms are dysopsia, dyslexia, sensory dyscopia.

(e) *Verbal Parasurdité* (Word Deafness). The principal symptoms being dysacusia, dysechographia, sensory dysechophemia.

(f) *Conduction Aphasia.* The vitiation of the communication between two centers.

(g) Examination of sensory or motor area corresponding to the wounded center.

Such is the bare outline of the author's scheme of examination.

2. *Late and Senile Epilepsy.*—Epilepsy does not spare the adult, and the relation, if any, existing between a late epilepsy and one of senility, is the thesis of the author. Two histories are detailed. Epilepsy occurring at the ages of 64 and 55 years respectively. In both patients there was an arterio-sclerosis, which the author holds is not the constant anatomical substratum underlying senile epilepsy, although it is widely recognized that arterial disease is the appanage of old age.

In an analysis of 1,196 subjects, those over fifty years of age were selected; of these there were 32, 12 being over sixty. This rarity does not seem to accord with the great amount of arterio-sclerosis at these ages. On the contrary, it would appear from the analysis, that hereditary predisposition seemed to occupy the most important position. The author does nôt agree with Maupate's dictum that after twenty years of age the hereditary rôle is unimportant. He believes he has seen cases that might be termed tardy epilepsy, in patients of from twenty-five to thirty-five years of age. The authors report but three cases to the general number. In all the family history was neuropathic, and in none did arterio-sclerosis seem a dominant factor.

3. *Suicide and Madness.*—The author continues his observations, speaking of the influences of imitation as an important element in the etiology of suicide. The article will be abstracted in full at its close.

4. *Language among Idiots.*—In this connection written language and mimicry are considered. The article is further continued.

5. *Protection of Fortune of the Insane.*—The laws bearing on this subject in various countries are given and discussed. It is a continued article, and its main deduction will be given at its close.

JELLIFFE.

Rivista di Patologia Nervosa e Mentale.

(Vol. vii, fasc. 5, May, 1902.)

1. A Case of Partial Epilepsy. G. Catòla.

1. *Partial Epilepsy.*—A description of a case in which operation confirmed the clinical diagnosis of brain tumor. The author's conclusions drawn from the literature of the subject are as follows: (1) the aura is the expression of a cortical irritation and represents the initial phase of the convulsive seizure; (2) the sensory-motor aura is the symptomatic expression of a stimulus which acts in the territory of the psycho-motor zone; (3) the sensory aura is the expression of a stimulus which acts outside of the psycho-motor zone and constitutes an important aid to the diagnosis of the site of a lesion located in the corresponding sensory centers or in neighboring parts; (4) as the aura is, for the most part, the result of a superficial and not very intense stimulus, it probably furnishes a more definite indication of the diseased site than the convulsion itself which is the result of a more intense stimulus and which carries with it phenomena of diffusion more or less extended and accentuated; (5) in rare instances a lesion capable of provoking convulsive phenomena is located exclusively in the white matter; in these cases it is impossible, during life, to make a differential diagnosis from the strictly cortical forms; (6) of great import is the site in which the convulsion begins, as well as its mode of diffusion; (7) Jacksonian epilepsy is not always the expression of a circumscribed cerebral lesion, but may accompany very diffuse lesions; (8) there are forms of Jacksonian epilepsy, which are due to extra-cerebral stimulus; (reflex forms) as well as those which are purely neurotic (hysterical forms); (9) other forms are due to auto- and hetero-intoxication, in which a lesion of the central nervous system is sought in vain; (9) nothing is known of the site of the cerebral lesion in masked partial epilepsy. (Equivalents.) R. L. Fielding (New York).

Brain.

(Vol. 25, 1902, No. 97, Spring.)

1. On Concussion of the Brain in Some of Its Surgical Aspects. Herbert W. Page.
2. Analysis of 155 Cases of Tabes. Byron Bramwell.
3. A Case of Congenital Muscular Atrophy (Family Type) and a Case of Hemorrhage into the Spinal Cord at Birth giving Similar Symptoms. C. E. Beevor.
4. A Brief Report of the Clinical, Physiological and Chemical Study of Three Cases of Periodic Paralysis. John K. Mitchell, Simon Flexner and D. L. Edsall.
5. Observations of a Case of Convulsions (Trunk Fit or Lowest Level Fit?). J. Hughlings Jackson and H. Douglas Singer.
6. On the Supposed Reversal of the Law of Contraction in Degenerated Muscle. W. Page May.
7. Internal Hydrocephalus in the Adult, with Remarks on the Etiology of Hydrocephalus and its Occasional Association with other Abnormal Conditions of the Central Nervous System. F. Parkes Weber.

1. *Concussion of Brain.*—In an exhaustive and highly practical presidential address the author considers the surgical aspects of cerebral concussion. At the present time the surgeon is more active in his operations in and about the brain cavity than formerly, and he holds that surgery may be even bolder than it has hitherto been in endeavor-

ing to minimize and remove some of the causes which seem to be at the root of many of the later consequences of severe head injury, especially in cases where accurate diagnosis may be impossible and the surgeon has to rest content with speaking of them as concussion of the brain, uncertain as to the precise injury which has been inflicted. Obvious grounds for surgical interference are the elevation of depressed bone and removal of fragments which have been driven into the the brain substance, it being constantly borne in mind that injury to bone and the deeper table of the vault of the skull may be more extensive than outside appearances suggest. Wounds of this nature in which the scalp and the bones are extensively injured do not provide he thinks the worst varieties of head injury. The traumatic opening itself seems to relieve intracranial tension, a supposedly unfavorable feature. Compression of the brain from meningeal hemorrhage is another obvious reason for surgical procedures. In the same category are to be grouped laceration of the sinuses, lateral or longitudinal. Still other cases offer some attractions. He then discusses some of the more important symptoms. Convulsions of the Jacksonian type. These are rare and are usually the result of a circumscribed local lesion. General clonic convulsions, however, are not infrequent. Severe persistent pain of a peculiar differential type, elevation of temperature, being noticeable in a large proportion of the author's cases which terminated fatally, 103°-106° F. without suspicion of sepsis, being recorded.

The author believes it justifiable in cases in which the symptoms seem to point to meningeal hemorrhage to trephine the skull and make a diagnosis, believing it to be a better procedure than Tuffiers' lumbar puncture. In the matter of brain disorder following cerebral concussion, the author is inclined to lay considerable stress upon cerebral concussion as a cause for epilepsy and insanity. Operative procedure may offer some hope in the former condition, but it is of very undecided value in the latter.

2. *Tabes.*—The author here analyzes 155 cases of tabes dorsalis. Of these 77.4 per cent. were in the ataxic and 22.5 per cent. in the preataxic stage. One hundred and forty were males, 15 females. The age at onset between 20 and 30 years, 79; 30-40 years, 77; 40-50 years, 35; 50-60 years, 18; over 60 years, 2. Thus 74.1 per cent. began between the ages of 30 and 50. One case began at 21 and there was no syphilitic history; another at 73. The age at onset of 565 cases of Bonar, Thomas, Riley and Bramwell 12.9 per cent. between 20-30; 43.9 per cent. between 30-40; 31.4 per cent. between 40-50; 11.6 per cent. above 50. In 90 the patients were married; occupations were various. Under etiology 56.7 per cent. give a definite history of venereal sore; 27.7 per cent. seemed free from syphilis; 167 per cent. of these had had gonorrhea. In 40.5 per cent. the disease developed within ten years of syphilitic infection; 59.3 per cent. more than 10 years; in 10 cases the time was more than 20 years. Sexual excesses were not numerous. Marriage in these with histories of sexual excess seemed to aggravate the condition. Alcoholic excesses were present in but a small proportion, 10; injury was marked in 6 cases; exposure to cold and wet, 6; mental worry, excessive standing, scarlet fever, diphtheria, each seemed related to one or two cases. The author concurs with the general opinion that syphilis is the most important cause of tabes. It is distinctly not the only cause, he holds. Three main factors always need considering: (a) the original constitution of the nervous system; (b) syphilis; and (c) cord strain or irritation. As to the mode of onset in 152 this was gradual. The early symptoms were pains, 82 cases; diplopia, 73; ataxia, 13; loss of vision, 9; pains and ataxia, 6; bladder, 6; gastric crises, 5; numbness of feet, 3, etc. The

author then analyzes the different symptoms. Lightning pains were present in 148 patients; pains in the back, 45; girdle sensation, 67; paresthesia, 103; anesthesia of legs, 68; of arms, 20; of trunk, 40; in face, 2; analgesia in legs, 55; in arms, 40; ulnar analgesia, 29 in 60; Biernacki's sign, 14 of 60; analgesia of trunk, thorax and abdomen, 59; thoracic analgesia, 45 in 60; analgesia in face and head, 2; heat and cold, few; ataxia in gait, 120; Romberg's sign, 120; incoördination of legs, 117; incoördination of arms, 39; muscular sense in legs, 67; in arms, 24; muscular analgesia in legs, 38; muscular hypotonia, 38; knee-jerks lost in 127; in 20 the knee-jerks were present; 5 exaggerated; ultimately 132 lost knee-jerks; Achilles jerk, 44; absent in 52 cases; deep reflexes 23; upper extremities, jaw-jerk, 7; plantar reflex, 19; Babinski sign, 4; 3 showed no movement; 39 flexion, and 5 extension; cremasteric reflex, 30; abdominal reflex, 55; bladder reflex, 115 affected in some way; rectal reflex, 98; sexual reflex, 73 affected, 72 lost, 1 exaggerated; paralysis, 16; pupils, unequal in size in 54; 39 equal; contracted in 69, medium in 27, dilated 8; pupil reflex to light, lost in 121, 24 brisk, 116 sluggish in both sides, 5 on one, ultimately lost in 145; accommodation reflex, 13 absent, 132 present; Argyll-Robertson, 90, ultimately in 112. Bramwell gives no support to the theory that the Argyll-Robertson is present only in those cases with syphilis; optic atrophy, 33; crises present in some form in 24, trophic lesions in 9; mental symptoms more or less typical of paresis 16. The results show that 31 have died, 75 still live, 49 lost to observation. Of the 75 still alive, 16 are distinctly better, the remaining 59 are either worse or in *statu quo*. Causes of death various. Duration of fatal cases average 8.3 years; longest, 22 years; shortest 1½. This is a most interesting and valuable summary.

3. *Congenital Spinal Muscular Atrophy and Spinal Cord Hemorrhages.*—Dr. Beevor describes two patients admitted about the same time to the hospital, both of which presented almost identical clinical pictures with very dissimilar causative agents. There was complete paralysis of all the muscles of the lower limbs and trunk, excepting the diaphragm; in the first case there was also complete paralysis in both arms, and in the second partial paralysis of one arm. In both cases all the affected muscles were flaccid and did not react to faradism, and there was loss of sensation in the legs and trunk as high as the 2d dorsal segment. The symptoms in the first case were due to a progressive atrophy of the cells of the anterior horns and a degeneration of the posterior columns which had commenced *in utero*, and in the other to hemorrhage into the spinal cord caused by dislocation of the spinal column at birth, and to stretching of the right bronchial plexus.

4. *Family Periodic Paralysis.*—A clinical, physiological and chemical study of three cases of family periodic paralysis is contributed by J. K. Mitchell, S. Fexner and D. L. Edsall. The paper shows in a fragmentary manner that the paralytic attacks are probably the result of some metabolic disturbance, perhaps situated in the muscles themselves affected.

5. *Trunk Fits.*—Dr. Hughlings Jackson and Douglas Singer report the history of a case of convulsive muscular movements analogous to an epileptic convulsion, but originating at levels lower than the cerebrum. Bulbo-pontal fits is a name suggested. The first symptoms were involvement of muscles of both sides of the neck, back and front, soon followed by fixation of both sides of the chest. The authors refer to the possibility of the involvement of Horsley's and Schäfer's trunk centers and to Sherrington's and Grünbaum's recently described Rolandic center. The fits are described very minutely.

6. *Reversal of Reaction of Degeneration.*—The author concludes from his study that Pflüger's law of contraction, according to which excitation occurs only at the point where the current is leaving all of the excitable tissue, is as true for degenerated muscle as for all other excitable tissues, and that the reversal of the law described by clinicians is only apparent, and is determined by the special imperfect modes of testing which have been in use.

7. *Internal Hydrocephalus of the Adult.*—Dr. Weber in a lengthy communication on this subject concludes: that (1) the various kinds of hydrocephalus and effusion into the ventricles of the brain may be fitly compared to the various kinds of effusions into the pleura and the peritoneum (2) the cases of so-called idiopathic or simple internal hydrocephalus are probably nearly all due to more or less localized serous meningeal or ependymal inflammation and are strictly analogous to cases of serous effusion into the pleura or peritoneum, resulting from localized non-supurative pleuritis or peritonitis. It is of course probable that there are several different microbic or toxic agents; (3) the reason why chronic inflammatory thickening of the membrane stretching from the cerebellum to the medulla and forming part of the roof of the fourth ventricle has so often been found present in fatal cases of chronic hydrocephalus, is not necessarily merely that the foramen of Magendie and the neighboring foramina have been closed by inflammation (as they very often must be in similar cases) but that this portion of the roof of the fourth ventricle is one of the sites of election for the localized inflammation which leads to hydrocephalus; (4) the theory of purely angioneurotic effusion to account for some cases of acute internal hydrocephalus has as yet not sufficient evidence to support it, though doubtless the amount of irritation required to produce the same pressure of effusion varies much in different individuals; it varies doubtless according to conditions (temporary or persistent, congenital or acquired) of the blood vessels and lymphatics which influence the local circulation; doubtless also temporary circumstances, such as exposure to cold or heat, the ingestion of alcohol or other stimulants, and reflex effects on the local circulation from other parts of the body, may influence the tendency to effusion in the ventricle; (5) many cases of apparently acute hydrocephalus in adults and children are really exacerbations of a chronic condition, sometimes, doubtless, dating from early childhood or birth, as evidenced by the history of previous cerebral symptoms by the relatively large size of the head, or by the post-mortem evidence of association with a condition of hydromyelia or syringomyelia, or, as in the present case, by excessive weight of the brain.

JELLIFFE.

Archives d. 'Electricité Médicale.

(No. 113. May, 1902.)

1. The Laws of the Transparency of Matter to X-rays and their Applications to Medical Radiology. L. BENOIST.
2. Lethargy, probably Hysterical in Character, and Its Treatment by Electricity. A. RIC and H. BORDIER.
3. A Phototherapy Apparatus in which the Iron Arc without Cooling Apparatus is Employed. A. BROCA and A. CHATIN.
4. The Medico-Electrical Installation in the Colonial Hospitals. DR. JOURDAN.
5. The Employment of Villard's Rectifying Interrupter for Producing X-rays and High Frequency Currents. DR. H. GUILLEMINOT.
6. A New and Simplified Light Bath with Incandescent Lamps. J. B.
7. The Utilization of 220 Volt Circuits to Actuate Röntgen Ray Coils. J. B.

1. *Transparency to X-Rays.*—By employing his *radiochronometer,* which has previously been described in these abstracts, the author has evolved the following laws relative to the specific transparency of matter to X-rays: (a) the specific opacity of a body for a given quality of X-rays and for a definite thickness of the standard is independent of the physical state of the body; (b) the specific opacity of a body is independent of the mode of grouping of its atoms or molecules; (c) the specific opacity is independent of the state of freedom or combination of the atoms. With the varieties of X-rays standardized by the author's method the classification, designation and rational usage of X-rays becomes a matter of precision.

2. *Lethargy and Hysteria.*—An account of a patient with recurrent attacks of hysterical lethargy which were characterized by an abnormal prolongation of the morning sleep, lasting on one occasion for twenty-two hours. As a result of numerous applications of a strong rythmically interrupted galvanic current applied to the neck and forehead, the duration of the attacks gradually lessened and normal sleep returned.

3. *Phototherapy Apparatus.*—A detailed description of an apparatus employing the iron-arc as the source of the violet and ultra violet radiations. In contra-distinction to the Finsen apparatus no intervening cooling cells are necessary as very little heat is radiated from the iron arc, which is inclosed in a metal chimney with suitable apertures for the exit of the rays and for observing the arc.

4. *Electrical Installation.*—A technical article.

5. *Villard's Rectifying Interruptor.*—A description of an ingenious and simple polarized device for rectifying alternate currents for actuating X-ray coils. A freely vibrating flat spring whose natural period of vibration approximately equals the period of the alternating current to be rectified is finally clamped at one end between the poles of a permanent horseshoe magnet. To the free end of this spring is attached a light metallic plunger which dips into a cup containing mercury. Surrounding the spring at a suitable point is a helix of wire which is traversed by the alternating current which thus alternately reverses the magnetic polarity of the spring. A detailed description of the various procedures necessary for the exact adjustment of phase is also added.

6. *Light Bath.*—A description of a light bath, made in the form of a frame composed of vertical tubes to which are attached the incandescent lamp sockets. The whole is covered by a canopy with an orifice at the top for the patient's head.

7. *Actuating X-ray Coils.*—A description of a rather unsatisfactory method for actuating induction coils on 220 volt circuits which consists in charging a large condenser directly from the 220 volt circuit and then immediately discharging it through the primary of the induction coil.

R. H. CUNNINGHAM (New York).

Revue Neurologique.

(Vol. 10, 1902, No. 8, April 30.)

1. Cerebrospinal Meningitis in the Form of Infantile Paralysis: Cytodiagnosis. RAYMOND and J. A. SICARD.
2. Infantile Spinal Paralysis Localized in the Muscles of the Superior Radicular Group of the Brachial Plexus. E. DUPRÉ and E. HUET.
3. A Particular Form of Relation of the Radicular Cells after Rupture of the Peripheral Nerves. G. MARINESCO.

1. *Cerebrospinal Meningitis and Infantile Paralysis.*—To recent works is due the review of acute infectious meningitis, the division into tuberculous and simple bacterial meningitis, and the classification of these according to the causal pathogenic agent. Two new methods, the bacteriological and the cytological study of the cephalorachidian liquid, obtained by lumbar puncture, have contributed to this end, and enable more exact diagnosis of the kind of case. Of the two methods the cytodiagnostic more frequently gives a positive response, more immediate and reliable than the bacteriological. A case is reported in detail of a child three and one-half years old, normal in every respect up to that age, who gradually lost appetite and became weak and ill, with symptoms of headache, painful vertebral column and articulations and contracture of muscles at the nape of the neck. After some improvement there occurred partial paralysis of the arms. Reaction of degeneration in certain muscles was determined.

Infantile paralysis seemed the diagnosis. True, brachial paralysis was present with well-marked radicular topography, but that was considered not unusual. At the advice of M. Jones, who made reservations in diagnosis and gave a hypothetical one of cerebrospinal meningitis, lumbar puncture was performed. A slightly cloudy liquid was obtained. On examination the percentage gave 80 polynuclear to 20 mononuclear cells. Second puncture, fifteen days later, gave a slightly clearer liquid with sufficiently abundant lymphocytes, the relation now being 70 mononuclear to 30 polynuclear. The diagnosis was altered to radicular brachial paralysis in the course of a cerebrospinal meningitis. Thus it was necessary to examine the etiological and pathological, if not specific unity of the meningeal and encephalomyelitic processes. One single pathogenic agent, pneumococcus or meningococcus, or still other microbes, may determine the disease by infection or by intoxication, either the localized lesions in the gray matter of the medulla, brain or roots of the medullary or of the peripheral nerves, or diffuse lesions in the different positions of the neurax. The idea of epidemicity, climate, age of the patient, primary or secondary localization of the meningeal affection are modifying factors of the virulence and vitality of the first pathogenic agent and may create diverse clinical types. Cytodiagnosis, examination of the cytological formula, quantitative and qualitative of the cephalorachidian liquid may give greater exactitude to the syndromes, still badly classified.

2. *Infantile Spinal Paralysis in the Brachial Plexus.*—Infantile spinal paralysis, paralysis and atrophy of the muscles may cause a disposition analogous to that in radicular lesions. A rare case is reported: a perfectly normal child of nineteen months with healthy parents was suddenly taken with a fever, but did not seem to suffer; after some days the right arm was noticed to be paralyzed; later the forearm hung in the habitual attitude of superior radicular paralysis of the brachial plexus; also the shoulder was slightly raised on the same side. Paralysis of the deltoid was shown by very pronounced distension of the scapulohumeral joint; finger movements were good. In electrical examination reaction of degenerescence was obtained in muscles of the Duchenne-Erb group. The child was regularly treated by galvanic electricity at the Salpêtrière. Infantile cervical paralysis was the diagnosis which immediately suggested itself; and the fact that in the inferior radicular group one muscle, the short abductor of the thumb, remained greatly altered, was in accord, and suggested acute anterior poliomyelitis. It may be asked whether there were not spinal or radicular alterations developed in the course of a cerebrospinal meningitis. Several interesting cases

of this nature are touched upon. The exact factor in diagnosis is given in lumbar puncture and examination of the cephalorachidian liquid; but this case came too late for that. Thus infantile paralysis is preferred to cerebrospinal meningitis as diagnosis, first on account of the precocity of the paralytic troubles, which are later in meningitis; also because of the absence of pain and stiffness of the side and neck; also the short duration of the paralysis in certain muscles.

If, as we believe, this was a case of acute anterior poliomyelitis, this observation furnishes a novel example of myelopathic muscular atrophy of radicular topography.

3. *Changes in Radicular Cells after Rupture.*—A type in this class of cells has been noted worthy of remark; the altered cell-bodies not only fail to present central chromatolysis, which is normally seen after the solution of the continuity of peripheral nerves, but on the contrary the chromophilic elements, sometimes massed and distributed concentrically about the nucleus, were strongly colored. Usually they form filaments more or less oblong in layers about the nucleus. The size of the filaments is variable, sometimes of short diameter, sometimes with longitudinal diameter scarcely exceeding transverse. Otherwise they look like long filaments. Illustrations are given with the article. Without the central zone was a peripheral arc, where colored corpuscles in the form of long filaments, much paler than those of the perinuclear zone, and of varied directions, were found; in the cell-body they were distributed in layers about the central zone; in the periphery the direction was longitudinal. In the protoplasmic prolongations the filaments were equally longitudinal, and notably one could see on some that not only in entering the cellular body did they keep their primary direction more or less independent of that shown by the corpuscles of the cellular body, but they crossed the cell-body to continue in protoplasmic prolongations nearby or on the opposite side. The special distribution of the chromophilic bodies of the protoplasmic prolongations following ablation of a more or less long nerve tract was found by the author, and the importance was hardly realized at first. The intimate relations of the tracts of fibrils of the achromatic substance with the corpuscles of Nissl was recognized; was this, then, a special reaction for ruptured nerve, as it has been noticed only in such cases? Rupture hardly explains it, as there was possibly a latent disposition brought into evidence by traumatism. After traumatism greater absorption of nutritive liquid takes place. This explains alteration in the volume of the cell; moreover, the fibrillar fascicles were more or less dispersed after serous discharge, so that the tract of chromophilic elements in the interior could be more easily seen. But this did not explain why the chromophilic elements were paler and less prominent in the periphery than in the central part. It may be admitted, the author says, from work done on the fine structure of cells of the spinal ganglia, that in certain cells the relation of the axis cylinder is closer to the peripheral fibrils, while in others the contrary is the case. JELLIFFE.

Neurologisches Centralblatt.

(1902. May 1, No. 9.)

1. Judges and Experts. NÄCKE.
2. Notice on Character Changes following Brain Injury. M. FRIEDMANN.
3. Further on Asthenic Paralysis. S. GOLDFLAM.

1. *Judges and Experts.*—A reply to Hoches' article (N. C'b't, No. 7) concerning the relations of the judge and the medical expert.

(2) *Brain Injury and Character Changes.*—Friedmann reports the pathological findings in two rabbits on which experimental puncture of the brain had been performed. A complete change in the character of the animals developed. This consisted mainly in a condition of ceaseless activity, and a change to a wilder disposition in the animals. In these animals there was present an intense lymphoid inflammation in the lymph spaces, about the blood vessels and in the pericellular spaces. These changes were very elusive and were considered as the manifestation of a condition of chronic irritation.

(3) To be continued.

McCarthy (Philadelphia).

MISCELLANY.

Alcohol a food or Poison? Kassowitz (Pfluger's Archiv. f. Physiol., June 3, 1902).

The conclusions of this author, as to whether alcohol can, in the same organism, act at once as a food and a poison, are as follows: There is a supposition that alcohol has a nutritive value, in that, during the process of its metabolism, a portion of it is directly split up or burned without being first built up into the protoplasmic substances. There follows, as a natural corollary to this belief the assumption that foods equally valuable as fuel can be substituted for one another. This, however, has been proved false by authenticated experiment. This hypothesis disposed of, there remains the reasonable supposition, borne out by the facts obtainable, that all foods are assimilated into the protoplasmic structures, the products of metabolism resulting from the splitting up of this chemical unity. A high degree of complexity of the protoplasmic molecule and unstability of equilibrium are implied by this assumption. The labile molecules of protoplasm are disorganized by all stimuli and chemically active poisons, therefore, since protoplasm is distributed by alcohol, not only as a stimulus but also as a poison, it follows that it cannot also act as a food. The toxic effect of alcohol is an increased nitrogenous elimination, the protoplasm disorganizing, either with the splitting off of fat and nitrogenous waste products, or a nitrogenous retention manifested by the splitting off and formation of gelatin-yielding tissues in the form of connecting tissue fibers, showing itself by the formation of inflammatory products in the different tissues and organs. After a short period of stimulation by alcohol the production of carbonic acid is decreased by paralysis of the centers of innervation and the consequent diminution of their activity, which does not mean a sparing of the tissue-fats and proteids, but is an indirect result of the toxic action of alcohol. The observation that a diminished working capacity and dissipation of the vital resources follows the substitution of alcohol of equal caloric power for a part of one's non-nitrogenous food, accords with the theory as stated. That the nutritive property of a substance cannot depend upon oxidation in the organism, and, therefore, that alcohol is useless as a food, is shown the author by these facts, and from them he draws the conclusion that neither for the sick nor well should alcohol be used for the purpose of supplying food.

Jelliffe.

Senile Tabes. A. Pitrés (Jour. de Med. de Bordeaux, May 18, 1902).

Although locomotor ataxia is most common between the ages of twenty-five and forty-five years, this author reports that one-fourth of the cases which he observed occurred after the fiftieth year, and of these three were after the seventieth year. Of these three, two had

enjoyed good health up to that time and the third had manifested tabetic symptoms after an attack of influenza. While the symptoms of senile tabes are generally the same as those shown in earlier life, the progress is usually more rapid and has no pre-ataxic stage. In two of the cases noted, autopsy showed marked atheroma of the peripheral and large spinal arteries, with atrophy of the posterior roots and sclerosis of the posterior columns, as in ordinary tabes. The author concludes that senile tabes is not so closely related to syphilis as that of early life, while it may be indirectly attributed to any acute or chronic cause inducing degenerative changes in the arterial system, as senility, alcoholism or infectious disease, and very likely constitutes a manifestation of spinal arterio-sclerosis, rather than an effect of luetic infection of the nervous centers. JELLIFFE.

CURE OF TUBERCULOUS MENINGITIS BY CREOSOTE. THOMALLA (Berlin. klin. Woch., June 16, 1902).

This author cites a case in which the use of creosote in generous quantities resulted in recovery of a patient suffering from tuberculous meningitis. In the treatment of the patient, reliance was placed on careful nourishment, the administration of potassium iodide and finally creosote. The subject of treatment was a young man of twenty whose parents had both died from tuberculosis, and who had already undergone cauterization for local tuberculous affections. The administration of creosote was begun at the commencement of the attack, the doses increasing from day to day until they reached the quantity of four and a half grams, administered in capsules three times each day. The diagnosis of this case was confirmed by Professors Von Michel and Gerhard. It would seem to be the opinion of several eminent practitioners that the drug should be administered in as large quantities as possible, inasmuch as it is rapidly eliminated. Thomalla limits the dose only at the point of toleration, and also calls attention to the fact that it is found in the blood serum in a like proportion. Others, among them Burlureaux and Chaunier, place the limit at five grams, the latter recommending it only in conjunction with outdoor treatment. It should be borne in mind that the efficacy of the use of creosote in the treatment of tuberculosis lies in its deadly effect upon the bacteria in the diseased tissues which it penetrates. JELLIFFE.

THE VOLUNTARY ERECTION OF HUMAN HAIR. S. S. MAXWELL (Amer. Jour. Physiol., July 1, 1902).

The erection of the hair, by the contraction of the arrectores pilorum muscles attached to the hair follicles, through the stimulus of fright or cold, which cause a bulging of the hair-follicles (goose flesh) is well known. The author cites a case of a patient who could at will erect the hair by voluntary bulging of the hair follicles. No edema or resemblance to factitious urticaria or the reflexes ordinarily seen in cases highly neurotic were present; neither was there change in color, with the exception of a slight pallor caused by the vasomotor change. The condition could be best observed on the arm, thigh, back and hips, and associated with it a remarkable development of the dermal structures was observed. The young man also had unusual control over the muscles of the ears and face. All these powers of control appeared to be obtained by inheritance rather than practice, for the subject's father also possessed them to a remarkable degree. Whenever he induced the phenomena the patient underwent modifications of the movements of respiration, the breathing grew deeper or ceased momentarily, and the chest felt as if more than usual in the phase of inspiration. The move-

ment gave relief from headache at times and was accompanied by sensations of pleasure; there was also dilation of the pupil of the eye, which would lead one to observe that the relation of vasomotor constriction and the dilation of the pupil was present here. It is the opinion of the author that in this case an unusual inherited neuromuscular and dermal development was displayed in connection with voluntary control of the arrectores pilorum muscles. In the lower animals the power of the erection of the hair would seem to be accompanied by a high development of the panniculus structures. In order to find out whether the arrectores pilorum contained striated muscles or not a biopsy was performed, but only smooth muscle was revealed. This discovery proved that the former distinction between voluntary or striated and involuntary or unstriated muscles could no longer hold good, as it was an example of smooth muscle which was at the same time voluntary. Where immediate and rapid action is required it would appear that striated muscles occur. JELLIFFE.

ACTION OF BATHS ON METABOLISM. H. WINTERNITZ (Deutsch. Arch. f. klin. Med., 1902, Vol. 72, Nos. 3 and 4).

The results of the studies of this author are stated by him as follows: (1) The sand-bath considerably increased the consumption of oxygen and caused a discharge of carbonic acid gas more than that occurring in fever, although there was no increase in temperature or change in the general conditions; (2) the mud-bath caused no especial change in oxidation; the application of irritants like mustard to the skin increased the production of heat and carbonic acid, and the consumption of oxygen; (3) the carbonic acid bath caused absorption of the gas; (4) the sulphur bath appeared to have no effect upon the exchange of gases. JELLIFFE.

HYSTERIA AND THE LACRIMAL REFLEX. W. G. SPILLER (La. Sém. Méd., 1902, No. 23).

From the observation of two patients the author suggests systematic tests for the presence or absence of the tear reflex when there is a failure of touch sense about the face, as a sure means of distinguishing between true centric and peripheral nervous disease and hysteria. The two patients upon whom these observations were made had had the Gasserian ganglion taken from one side for obstinate neuralgia. On tickling the nasal mucous membrane on the side of the operation, he found that no tears could be made to flow from the eye on either side, but if the mucous membrane of the nares on the normal side was touched, an immediate reflex flow of tears followed. When bits of paper were placed between the upper lid and the ball of each eye no tears appeared on the side from which the ganglion had been taken, but there was a copious flow from the opposite eye. The important differential point demonstrated by these experiments is that, while the manifestations of hysteria attack the eye quite as much as the other organs, chiefly in the form of anesthesia of the conjunctiva, and the palpebro-ocular reflex may be totally abolished, the lacrimal reflex is not affected at all. By the use of Dr. Spiller's experiments hysterical bulbar anesthesia can be readily distinguished from that due to trigeminal partial or total paralysis. JELLIFFE.

Book Reviews

Clinical Psychiatry. A Text-book for Students and Physicians. Abstracted and adapted from the sixth German edition of Kraepelin's "Lehrbuch der Psychiatrie." By A. Ross Defendorf, M.D., Lecturer in Psychiatry in Yale University. New York. The MacMillan Company.

The object of this work, as the author states in his preface, is at once to provide a text-book for his classes, and to make accessible to the English reading profession the teachings of Kraepelin in psychiatry. In this latter capacity a review of the work will necessarily involve, to an extent, a criticism of the German school of psychiatry of which Kraepelin is the acknowledged head.

The book is divided into two parts: the first dealing with General Symptomatology; the second with the Forms of Mental Disease.

The first part—General Symptomatology—takes up seriatim the disturbances of perception, mental elaboration, the emotions, volition and action. It is an admirable although not very exhaustive exposition of the subject, and shows a praiseworthy departure from the beaten paths in this domain. There is an evident attempt to call upon the modern conceptions of psychology to assist in the explanation of symptoms with a most happy result. The style is somewhat labored and cumbersome and to those unacquainted with German methods of expression there are many terms which at first must be confusing, such as "flight of ideas" and "pressure of activity."

The second part, devoted to a description of the Forms of Mental Disease, is divided into thirteen sections.

Section I gives a short and concise description of fever delirium and the post-febrile psychoses.

Section II is devoted to the exhaustion psychoses, collapse delirium, acute confusional insanity (amentia), and acquired neurasthenia. More space might with advantage have been devoted to this section, particularly to the descriptions of collapse delirium and acute confusional insanity. Confusional insanity has never received sufficient recognition in this country and alienists still go on, especially in state hospitals, classifying these cases as melancholias and manias and by so much distorting their statistics. It should have official recognition in all such institutions.

Section III is devoted to a consideration of the intoxication psychoses. The descriptions are good and for the most part conform to well established lines.

Section IV briefly considers the thyroidgenous psychoses. The author has purposely cut this short as having little practical importance for American alienists.

Section V on dementia præcox is one of the best in the book and evidently here the author has translated quite fully from the original as the descriptions are full and quite Kraepelinian. The clinical delineations of three forms, the hebephrenic, katatonic and paranoid are most admirable, and if they do not carry the conviction of the existence of such species we must at least acknowledge the grave significance of many of the symptoms upon which their diagnosis is based: *i.e.*, stereotypy,

verbigeration, negativism, muscular tension, emotional deterioration, the so-called katatonic stupor and the development of mannerisms.

Sections VI and VII on dementia paralytica and organic dementia respectively contain little worth special mention. The description of paresis is full but leaves the old saw as to its etiology *in statu quo*, whereas organic dementia is treated much too briefly.

Section VIII on the involution psychoses is noteworthy, particularly because of the restriction of the term melancholia to mental depression occurring at this time.

Section IX devoted to the manic-depressive insanity comprises with Section V on dementia præcox really the meat of the book. Kraepelin's teachings that all the manias and melancholias together with the various forms of circular and alternating insanities are but clinical manifestations of one and the same underlying condition, parts of the same disease, are only beginning to be noticed, but wherever these views have met with serious consideration conviction of their soundness has inevitably followed. The wonderful way in which difficult clinical problems are cleared up by this conception recommends it at once to all who have mastered it. Particularly is this so with reference to those conditions now known as "mixed states," which heretofore it has been impossible to classify in any of the niches of the psychiatric nosology.

Section X on paranoia is rather meager for so important a subject, only thirteen pages being devoted to its consideration. The erotic and religious varieties merely receive mention, while the original paranoia of Sanden (*originäre verrücktheit*) is not noted.

Section XI deals with epileptic, hysterical, and traumatic insanities. In spite of the fact that the author states in the preface that special stress has been laid on this section the descriptions of these several conditions are not over full. This is especially true of that part devoted to hysterical insanity. While the name of a German translation of one of Janet's works appears in the bibliography—"Der Geisteszustand der Hysterischen"—there is little in the text to indicate that the author has read it, and that most admirable later work by the same author, "Névroses et Idées Fixes," is not even mentioned.

Section XII deals with constitutional psychopathic states. Here we might expect to find a consideration of the délire des dégénérés of Magnan and the French school, but the condition is not mentioned. A rather forced distinction is made here between compulsive and impulsive insanity based upon the definitions in the first part of the book as follows: "Compulsive acts are those which do not arise from normal antecedent consciousness of motive and desire, but seem to the patient to be forced upon him by a will which is not his own."

"Impulsive acts are distinguished from compulsive acts, in that they do not seem to the patient to be influenced from without, but are the direct expression of a sudden overwhelming impulse, which gives no chance for reflection or resistance." The author acknowledges that these two forms are closely allied on page 391, where he says: "On the other hand, impulsive insanity approaches very closely some forms of compulsive insanity." It would seem that the French conception of these states as outlined by Régis was far preferable.

Section XIII contains a terse description of imbecility and idiocy. The imbeciles are divided into two clinical groups, the stupid and the active; while the idiots are described under the designation of severe and light forms. There is no attempt to classify them in accordance with the clinical types so well delineated by Ireland.

In conclusion we would say the first part—General Symptomatology —and the sections on dementia præcox and manic-depressive insanity, are most excellent and fully justify the existence of the work, rendering as they do the teachings of Kraepelin and the German ideas accessible to English-reading alienists, and we think no library in this special branch of medicine can be complete without either it or its German counterpart. WM. A. WHITE (Binghamton).

DIE OTITISCHEN ERKRANKUNGEN DES HIRNS, DES HIRNHAÜTE UND DER BLUTLEITEN. Von Dr. OTTO KÖRNER, Ao. Professor der Medecin, Director der Klinik und Poliklinik für Ohren und Kehlkopfkranke in Rostock. Mit einem vorwort von ERNST VON BERGMANN. Dritte Auflage. J. F. Bergmann. Wiesbaden, 1902.

This work covers in a very comprehensive and critical manner a field of medicine which is of the greatest importance and interest to the neurologist. It is true that he shares this field in common with, though perhaps to a somewhat less active degree, the otologist and yet our present knowledge of the conditions arising in the cranial cavity is so deficient that the combined efforts of both specialists are needed for its furtherance. As a contribution in this direction the work of Körner, whose authority is recognized the world over, must assume a high rank.

The book is an enlargement and revision of the second edition and contains much additional matter gained from a wider clinical experience and riper knowledge as well as from an exhaustive study of the literature of the last few years.

The first part is devoted to a consideration of uncomplicated disease of the meninges. Serous meningitis is recognized as occurring frequently in association with otitis and mastoid disease, and Körner even claims that it is often combined with an encephalitis. In treating otitis cases, where such complication is suspected, he urges that after operating upon the primary focus in the ear the surgeon should wait before proceeding to the dura in order to give the meningitis serosa time to subside, as it often does, and also thereby making possible a differential diagnosis from other forms of meningitis and intracranial disease.

In the section devoted to sinus disease Körner calls attention to the greater frequency of involvement of the right side in sinus thrombosis and extra-dural abscess, and explains this by the anatomical relations of the parts. Osteophlebitis or phlebitis of the veins of the mastoid process causing septic infection of the sinus, is a condition which he, first, really described and called to the attention of the medical world, and in this edition he maintains his views regarding its nature and substantiates them by additional clinical data. He also makes a distinction between pyemic and septic infection as it occurs through the medium of the sinuses.

In his operative procedure he recommends following the advice of Lane of exposing the dura of the middle and posterior cranial cavities when pus or cholesteatomata are present in the mastoid, instead of relying on symptoms for the diagnosis of intracranial conditions. He has never seen any harmful result from this step, and he has twice discovered and relieved unsuspected extra-dural abscesses. His consideration of the subject of brain abscess is very thorough and most instructive. In this condition he expresses the opinion that the chisel used vigorously undoubtedly causes a latent abscess to become active, or to rupture into the ventricles; a sinus thrombus to become loosened from the sinus walls and a localised meningitis to become diffuse,—facts which it were well to bear in mind.

Of especial interest to the neurologist from a diagnostic standpoint

are his conclusions as to the value of the lumbar puncture. He classifies the findings as follows: First, when a very much clouded fluid containing pus cells and bacteria is obtained, it means diffuse purulent leptomeningitis. An exception is in those rare cases where the suppuration is limited to a part of the vertebral canal, and which is not in relation to the meningitis of the brain. Second, a lightly clouded fluid containing bacteria can signify a diffuse or a circumscribed meningitis. An exception was a case of Briegers, in which an abscess in the temporal lobe led by a fistula into the ventricle and thus caused the lumbar puncture fluid to be purulent with the disastrous consequence that no operation was undertaken because of the presence of meningitis. He cites also a case by Wolff and Ruprecht where the puncture caused a similar faulty diagnosis.

The bibliography on each subject is very full and complete up to October, 1901. There is page reference to every case or author mentioned in the text, thereby doubly enhancing the value of this excellent treatise.

It certainly deserves translation into English, that it may serve as a guide to all who have to deal with cranial disease.

I. Strauss (New York).

News and Notes

Dr. A. P. Ohlmacher has been appointed superintendent of the Ohio State Hospital for Epileptics at Gallipolis.

Dr. F. M. Jelks has removed from St. Louis and has taken up the management of the Ozark Sanitarium on the death of his father, the late James T. Jelks.

Dr. L. Pierce Clark, associate editor of the Journal, has been spending the past year in Europe. He returns to take up practice in New York this fall.

Dr. Florence Hull Watson, first assistant physician of the Delaware State Hospital at Farnhurst, formerly in charge of the Pathological Department of the State Hospital for the Insane at Norristown, Pennsylvania, has resigned to continue work in Europe.

Dr. Ross Defendorf, of Middletown, Conn., has been spending the summer at Heidelberg, working with Professor Kraepelin and Nissl.

Miss Ella Welbor Cramer and Dr. John Joseph Kindred, of New York, were married at the American Church, Luzerne, Switzerland, at high noon, July 10, the civil ceremony having taken place in the Hotel de Ville, Luzerne, an hour earlier, in the presence of Mr. Morgan, United States Consul to Luzerne; Mr. Julius Hartmann, former United States Consular Agent; Mrs. E. A. Wheeler and several other friends of the bride and groom. The bride is a graduate of Vassar College and is popular in society and church circles in Poughkeepsie, N. Y., her native city. She is the only daughter of the late Mr. and Mrs. George Edward Cramer, the latter having been one of the most influential and public spirited citizens in Poughkeepsie, and a member of the firm of Reynolds and Cramer of that city.

Dr. Kindred is a native of Virginia, but for some years has been a prominent physician and specialist in New York City. He is also president and consulting physician of the River Crest Sanitarium for nervous diseases at Astoria, Long Island, New York City.

The last number of the *Rivista Sperimentali di Freniatria*, vol. 27, fas. 3, 4, contains an excellent portrait of Dr. A. Tamburini, with a short dedication and notice as a tribute to the twenty-fifth anniversary as editor of this excellent Rivista.

Doctors G. Cesare Ferrari and Arturo Donaggio, of the Psychiatric Institute of Reggio, have obtained the title of free docents in clinical psychiatry at the University of Modena.

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Original Articles.

A STUDY OF LANDRY'S PARALYSIS; WITH A REPORT OF THREE NON-FATAL CASES.[1]

By Theodore Diller, M.D.,
Pittsburg.

Ever since Landry, in 1859, gave the account of a patient who exhibited a certain set of symptoms and whose illness ran a certain course, there has been an increasing number of more or less similar cases reported bearing his name. The most generally accepted definition of Landry's paralysis seems to have been that given by Leyden and quoted approvingly by Thomas[2], that it is a rapidly ascending motor paralysis of sudden onset, with loss of knee-jerks, with slight or no sensory symptoms, without atrophy, loss of control of sphincters, electrical changes, or mental involvement and which runs a rapid course without fever and terminates fatally, usually exhibiting bulbar symptoms near the end. Mills and Spiller[3], writing more recently (1898), understand Landry's paralysis to be: "A disease in which rapid loss of motor power usually begins in the lower limbs, and the paralysis is flaccid and associated with paresthesia and loss of tendon reflexes. The upper extremities are soon involved, and bulbar symptoms develop after a few days. Pain is not a prominent symptom. Death occurs within a week or ten days, though in some cases it may be delayed, and in

[1] A paper presented at a meeting of the Pittsburg Academy of Medicine, held Sept. 29, 1902.

others, more rare, recovery may ensue. There may be a descending as well as an ascending form of paralysis. The electrical reactions are normal and patients are perfectly conscious of their condition." Thus it will be seen that a descending type is allowed, non-fatal cases are admitted, and paresthesia is considered a constant symptom although severe or persistent pains are not accounted as belonging to the clinical picture. Absence of marked elevation of temperature and non-involvement of the sphincters are by most writers named as distinguishing marks of the affection although not included in Mills and Spiller's definition. Landry's post-mortem findings were negative as were most of those of the earlier investigators.

The literature of Landry's paralysis has now become quite extensive; and in the several papers by Diller and Meyer[4], Bailey and Ewing[5], Mills and Spiller[6], Thomas[7], Knapp and Thomas[8], Taylor and Clark[9], Raymond[10], Oppenheim[11], and Schwab[12], will be found references to almost all the reported cases of this disease up to the year 1901, together with critical studies based upon them. A review of this literature, in a most striking manner, bears witness to the strong reverence which exists in the medical mind for a literary tradition, and the great hinderance which it may prove to a proper study of a disease. After forty years, during which many scores of reports of Landry's paralysis have been made, it remained for Taylor and Clark[13] to entirely break away from the literary tradition of Landry's paralysis in these forcible words: "On the basis of an acute, clinical, and necessarily highly superficial, pathological observation made many years ago by Landry, subsequent observers, led largely by a name, have been attempting to force into the narrow clinical conception outlined by Landry, various symptoms, none of them fundamental and supported by no constant pathological findings, and still less by a definite etiology. The so-called disease has with each new investigation lost something of its coherence. We have persistently clung to a name and demanded that the symptoms which it represented should conform to our preconceived idea." These views of Taylor and Clark are endorsed by a later writer, Schwab[14], and are to my mind none too strong. Although some recent

writers enlarge upon Landry's original definition and recognize "transitional forms," they have practically accepted the older idea that Landry's paralysis is a disease entity. Even such an acute observer as Dejerine[15], in his recent work, follows Landry in his description almost slavishly, accounting rapid ascending course, loss of reflexes, absence of marked sensory symptoms, absence of fever, preservation of sphincters and a fatal termination as marks of the disease. Schmaus[16], in his book published only last year, adopts the same views.

In the various attempts to study Landry's paralysis, the general plan has been to reject all cases which did not pretty closely conform to the type Landry described, or which did not come to autopsy, while but little attention has been paid to the etiology; and the non-fatal and atypical cases have been thrown out of consideration. If this plan were continued, the original Landry's paralysis clinically would, of course, be preserved despite the variable symptomatology and the conflicting post-mortem findings. Indeed, Goebel[17], writing in 1898, practically makes a plea for this method of study and deplores the tendency to bring many types under the head of Landry's paralysis.

While the various post-mortem studies have added much to our knowledge of Landry's paralysis, if not offering a full explanation of it, and are most praiseworthy, it occurs to me that too little attention has been paid to the clinical side, especially to the study of non-fatal and atypical forms of the affection. For it will no longer do to throw out cases which do not terminate fatally, or which depart from the classic type as described by Landry. It would be as reasonable to establish a pathologic basis by throwing out all findings which do not fit a certain type, as to insist upon such a clinical basis; and the pathologic studies have certainly revealed one thing, *viz.:* that there is no constant anatomic basis for the disease, even for the typical cases.

Let us now look at some of the clinical reports with the view of determining the range of symptomatology of the affection, noting especially those clinical manifestations which represent departures from the older definition of the affection.

Bailey and Ewing, in their elaborate and painstaking study

in 1896, collected all the cases which had, up to that time, been examined post-mortem, 75 in number. But of these 31 were thrown out of consideration either because post-mortem examinations were incomplete or unsatisfactory, or else because the cases were atypical clinically. Cases of Bernhardt, Pitres and Vaillard, Hayem, Pribytkow and Brochain, because of the presence of marked sensory phenomena; the cases of Vierordt, Strümpell, Eichorst, Diller and Meyer, because the course was prolonged or chronic; while Roth's case is excluded because the paralysis was not ascending in type. After this weeding-out process, Bailey and Ewing consider 44 of the 75 collected cases. In 12 out of 32 where the symptom is recorded, the sphincters were involved *i.e.*, in over one-third of the number analyzed. The knee-jerks were lost in 31 cases, diminished in 2 and increased in 2. Of 20 cases the electrical reactions were normal in 19 and lost in only 1. The temperature was normal in 20, while 14 exhibited moderate fever. As to sex there were 29 men and 10 women. In 3 cases the paralysis affected the arms and legs simultaneously; while in the remaining cases it began in the legs, in 4 cases in one leg before the other. There was sensory involvement in 13 cases.

Thus it will be seen that after excluding a large number of cases because they did not fit into the classic clinical type that certain atypical features are present in those admitted, *e.g.*, sensory involvement in one-third and sphincter involvement in more than a third of the cases. In 3 cases the paralysis was not ascending but began in all four members simultaneously.

Thomas[18] reports 2 cases which came to autopsy in both of which degeneration of the anterior horn cells and peripheral nerves was discovered. Thomas holds that Landry's paralysis is an affection of the lower motor neurone, and that the sensory apparatus is uninvolved. He mentions, however, that there was tenderness on pressure in one of his cases, and of the other that the patient's chief complaint on entering the hospital was numbness of the legs.

In Goebel's case, weakness of the vesical sphincter and diminution of sensation are noted as symptoms which appeared late in the disease.

Of Knapp and Thomas'[19] three cases only one was fatal and in this case there was some affection of the sphincters and moderate sensory disturbance. In one of the non-fatal cases, there was loss of control over the anal sphincter, sensory disturbances and muscular atrophy. The second non-fatal case also exhibited muscular atrophy.

In a case reported by Burghart[20] electrical reactions of degeneration were observed. This patient recovered somewhat, but finally died at the end of 14 days.

In a fatal case recorded by Robinson[21] numbness of the feet was the first symptom; pain in the back was complained of and the legs were tender when grasped.

In the fatal case recorded by Taylor and Clark[22] pains were present for a month before motor symptoms appeared. Pains in the back and dorsal portions of the thighs were at times extreme. Marked fibrillary tremors formed part of the clinical picture. Pressure over the nerve trunks produced no pain. Clinically this case resembles pretty closely one of my own.

Mills and Spiller state, of the case they record, that some weakness was present for a month before the actual symptoms of the disease appeared. Numbness of both hands with some pain and inability to perform finer movements were noted. Power left the arms first.

Worcester[23] mentions loss of sensation in the legs as a symptom in his case. Numbness in the hands with some loss of the finer movements were premonitory symptoms. The patient was mentally apathetic. There was rapid motor and sensory involvement.

Marie and Marinesco's[24] patient suffered intense pain over the entire body although a microscopic examination showed the peripheral nerves to be normal.

Hirtz and Lèsnè[25] describe a case which was at first acute and then chronic, and which terminated fatally at the end of four months. The muscles of the lower extremities were very tender and the seat of severe pains, increased upon movement.

A. Schultz[26] records two fatal cases. In the first case, the symptoms were those of acute ascending neuritis. In the second case the symptoms followed multiple neuritis in an alcoholic subject.

A case described by Oettinger and Marinesco[27] presented much objective diminution of all forms of sensation.

In a case recorded by Howard [28] paresthesia was the first symptom, and tingling and numbness were very distressing. The muscles were affected in the following order: bulbar, hip, thigh, shoulders, arms, legs, hands, feet, labial, diaphragm.

Spiller[29] reports a typical case in which improvement set in after two or three weeks and progressed to a complete recovery. Nearnow[30] also reports a case in which recovery occurred. Rowden[31] reports the case of a boy aged 10 years in which the paralysis was of a rapidly descending type beginning with headache, pains in the neck, a temperature of 103, and terminating fatally in a few days.

In Schwab's[32] case, which terminated fatally at the end of seven weeks, there was paresthesia in the fingers of the left hand for a week before the onset of the paralytic symptoms. There was marked hyperesthesia of the abdominal walls.

Wappenschmidt[33] reports a case of sudden onset attended with pain which ran a rapidly fatal course. The peripheral nerves were found normal although the clinical signs had pointed to a peripheral neuritis.

In a fatal case reported by Griffith[34] in a man aged 32, there were weakness and malaise for a week, then severe pains in the legs followed by a rapidly ascending paralysis which became complete. The diaphragm was paralyzed. There was some blunting of sensibility. The sphincters remained intact.

Paresthesia of the hands and vertigo were for eight days premonitory symptoms in a fatal case reported by Kapper[35]. Then headache and pains in the legs appeared, followed by rapidly ascending motor paralysis involving the bulb, laryngeal palsy occurring; and death ensued. The knee-jerks were lost. Consciousness was preserved intact to the end. In the beginning of the attack hysteria was considered. Although the cord and peripheral nerves were examined by the Nissl and osmic acid methods no pathologic changes were discovered in them. A bacteriologic examination was likewise negative.

Soltman[36] notes a case of Landry's paralysis in an eleven-year-old girl, where recovery occurred following treatment by inunctions.

Stockton[37] reports the case of a girl aged 27 (diagnosed acute ascending myelitis), where, following premonitory symptoms consisting of nervousness, insomnia and vague pains, which had existed two weeks, pain in the back and abdomen and anesthesia in the gluteal region developed. Control over the bladder and bowels was lost. Paresthesia in the feet developed along with weakness in the legs which, two days later, were completely paralyzed and anesthetic. The next day the arms were almost completely paralyzed and sensation in the upper part of the body was much impaired. Some movement in the legs now returned. The knee-jerks were absent. The neck muscles became involved and the patient died, being conscious to the end. No autopsy was held. Hematoporphyrinuria was a persistent symptom, although only very little trional had been given.

Maxwell[38] records the case of a Chinese woman in which the paralysis was of an ascending type, involving the bulb, and which ran a fatal course in 12 days. Paresthesia in the feet and pains in the legs preceded the motor involvement. The knee-jerks were absent and the urine passed involuntarily. Just before this attack the patient had suffered from diarrhea produced by eating unripe fruit.

In a case examined post-mortem by Wappenschmidt[39], the peripheral nerves were found to be normal. The anterior horns and Clarke's columns showed degeneration of increasing intensity from below upwards. Dydynski[40], on the other hand, holds that Landry's paralysis is a peripheral nerve disease and ascends because it attacks the least vital parts first, these being least resistent to toxic influences. To support his view he points to the effects of curare poisoning upon animals.

Baumstark[41] reports the case of a young man (which he regards as one of ascending acute myelitis) who had contracted malaria in Africa. He suddenly developed weakness in the legs which rapidly increased to complete paralysis. Motor weakness ascended; marked bulbar symptoms appeared. Muscular atrophy, absent knee-jerks, pains along the nerve trunks, produced by pressure, disturbance of sensation, paralysis of the bladder, impaired vision, and peculiar choreiform movements

of the hands were among the symptoms noted. The symptoms all gradually disappeared and recovery occurred. This case is of peculiar interest in that malaria seems to have been the cause, and because of the recovery after such a profound involvement of the nervous system.

At this point I shall introduce the clinical histories of three cases I have observed:

Case I is that of a single man aged twenty-one years, a telegraph operator, of excellent habits. He has never used tobacco or alcohol and denies syphilitic infection. The sisters died of phthisis; otherwise his family history is good. The patient himself had an attack of typhoid fever at the age of 14. Since then, up to the time of his present illness, he has enjoyed excellent health.

Four weeks ago, while telegraphing, he lost some control of his finger-movements in operating his instrument so that he became clumsy and awkward. He thought of writer's cramp. Four or five days later he became weak in the legs so that his knees often gave away in walking. At the same time his thighs and legs, especially the latter, felt numb. A week later the same numbness appeared in his arms, hands, and abdomen. He felt as though a band were about his waist. He now saw double at times—always when looking to the right or left or downwards. He also became dizzy on sudden movement. There had been no pain or fever at any time.

Examination on January 31, 1902. Stripped, he exhibits no atrophy. His gait is slow, labored, one of weakness. The grasp of the hands is feeble. There is no objective disorder of sensation; but he complains of paresthesia in arms, legs and abdomen. There is no pain upon pressure over the nerve trunks. The knee-jerks and Achilles jerks are absent. The abdominal reflex and wrist-jerk are present. The electrical reactions of the muscles of the arms and legs are normal. Eyes: The pupils are 3 mm wide, equal, and respond promptly to light and in accommodation. The eye-grounds are normal. In conjugate movements there is some muscular deficiency noticeable especially in movement to the right. No squint is apparent. The urine is normal.

He was observed only once; but a month later he was seen walking on the street. His sister stated that he took great aversion to some mutton he ate two weeks before the appearance of the first symptoms; said it was tainted and had spoken much about it.

Case II.—A perfectly healthy boy, aged 7½ years, went to

bed in his usual health. The next morning he complained of soreness in his legs and thighs. But little attention was paid his ailment and he attended school as usual. The pains or soreness continued; and three days later appeared in the arms and shoulders. At the same time it was noted that he had difficulty in walking. He was unable to return from school and had to be carried home. Very soon afterwards loss of power appeared in the arms; and three days after the onset of the paralysis he was unable to stand alone and his hand-grasp had become almost nil. He had insufficient strength to shut a door, but was still able to grasp a pencil firmly enough to write his name. He could not move his toes. His condition remained thus for about one week and he then began to improve and has steadily continued to do so ever since. There was no fever at any time; no mental, bulbar or sphincter symptoms. The pains complained of were never severe. He spoke rather of soreness and never cried because of it.

Examination, April 22, 1902 (five weeks after the onset of the trouble). The boy has a waddling, non-spastic gait. The grasp of the hands show about one-half the normal strength. There is no apparent wasting of the muscles (father notes none). The calf and thigh muscles are firm; but the arm and shoulder muscles are flaccid, soft and exhibit R. D. The leg and thigh muscles exhibit partial R. D. Sensation is unimpaired. The knee-jerks are present although somewhat sluggish. On deep pressure there is moderate tenderness in the arms and legs. The boy is quite bright mentally. Since the examination the boy has steadily improved, but is not yet well.

Case III.—A single man, aged twenty-five years, born in Ireland, a laborer. Family history negative. The patient had always been a hard worker and enjoyed good health.

The latter part of October, 1901, three weeks after coitus, a single, hard, non-painful sore appeared on the head of the penis. Following this he had a sore throat which lasted seven weeks. He also noted swelling of the glands in the groin. No skin eruption appeared. About the middle of November (three weeks after the appearance of the chancre), he was seized with severe pains in the legs; and 24 hours later with pains in the lower part of the back; and 48 hours later, with pains in the arms. These pains have persisted, night and day ever since. He had no pains in the trunk or head at any time. The pains have been more severe and more persistent in the legs than in the arms. He describes them as "steady," "sharp," "burning" pains. They do not leave or return suddenly, but are more or less constant, but always worse in the mornings. Along with the pains, he complained of tire, weariness, loss of strength.

From the time of the onset of his symptoms up to the date of his admission to the Mercy Hospital, January 23, a period of about nine weeks, he lost 23 pounds in weight. The pains have kept him awake much at night.

Examination, February 1.—There is general emaciation. A dusky reddish color of the skin over the arms and legs is noted. The pallor produced by finger pressure disappears very slowly. Fibrillary twitchings in the face, arms and legs are present. The twitchings are most marked in the face about the mouth and chin. Mentally the patient is quite clear, but gloomy as to the outcome of his trouble. The grasp of his hands is pretty fair, but probably much below what is normal for him. The legs exhibit as much or rather more weakness than the arms; but he can still stand for a short time on one foot alone. There is no swelling or pain about any of his joints. Deep pressure over the great nerve trunks produces no pain. There are no cranial nerve palsies. The pupils are equal (3 mm) and react promptly to light. There is no diplopia. The tongue is clean. Both knee-jerks are very quick, but make a rather limited excursion.

There is general glandular enlargement, the epitroclear, inguinal, and post-cervical glands being clearly palpable. A careful examination of the thoracic and abdominal organs by Dr. Johnson was negative. The pulse is rapid (100). There is no fever; and there has been none since he entered the hospital. A blood examination revealed the following: White cells, 10,720; red cells, 5,100,000; hemoglobin, 85 per cent. The patient has, at all times, had control over his bladder and bowels.

March 3.—The pains are considerably less in severity but still quite troublesome. He is more emaciated than he was a month ago. The bony points about the pelvis have become very prominent; and there are two or three small shallow bedsores over the sacrum. He walks in a feeble, laborious fashion with his legs partially flexed. The knee-jerks are now practically absent, the right responding but faintly. Achilles jerks are also absent. Deep pressure over the nerve-trunks and passive joint movements elicit no pain. The fibrillary twitchings especially in the legs are more marked than at the previous examination. [He had been walking about the ward just before I came and states that this always increases the twitchings in his legs.] On several occasions during the past month he has been unable to empty his bladder.

By way of treatment he received iodide of potassium, hydrotherapy, and remedies for the relief of pain and to promote diuresis.

All three of these cases are atypical and I dare say there are those who would hesitate or decline to classify them as Landry's paralysis, and yet all of them, I believe, exhibit a sufficient number of the marks of that affection to be so designated. Moreover, the clinical pictures which they present could not well be labeled by the name of any other disease.

These three cases are alike in that they all exhibited a rapidly ascending motor paralysis without elevation of temperature or mental symptoms. In all of them sensory symptoms were present, which in two of the cases preceded the motor symptoms; none had a fatal issue.

Case I is noteworthy inasmuch as the course of the affection was comparatively mild, the patient probably never having been off his feet.

The second case must, I think, be admitted to be a fairly typical one. Here, after three days of soreness in the legs and thighs loss of muscular power suddenly developed in these members, followed very shortly by similar involvement of the arms. This rapidly developed until it became almost complete; remained thus stationary for a week and then rapidly improved. Two interesting features of this case are the preservation of the knee-jerks, although diminished, and the presence of complete and partial reactions of degeneration in the affected muscles five weeks after the onset of the trouble. In neither of these cases was the cause of the disease clear.

The third case, on the other hand, is striking in that it seems almost certain that syphilis was the etiologic factor to which the disease was chargeable. In this case the sensory symptoms at first overshadowed the motor, making it seem like an ascending sensory rather than ascending motor disease. But along with the sensory involvement there were also motor symptoms, although in the background at first. It is to be especially noted that the involvement was rapid and ascending in type. Other points of interest in the case are the great and progressive muscular wasting; marked fibrillary twitchings involving the entire body, including the face; the, at first, exaggerated and afterwards absent knee-jerks; the vaso-motor disturbances; the slight involvement of the

sphincters; absence of fever; apparent absence of blood-changes; absence of mental symptoms; absence of bulbar involvement; the preservation of sufficient strength to walk and give a fairly firm hand-grasp; the absence of pain on pressure; the long and chronic course of the disease.

In one of the cases described by Landry there was a history of syphilis; and the possibility of syphilis as an etiological factor is considered by him. Nonne[42] believes that it is improbable that syphilis can produce the symptom-complex of Landry's paralysis and leave behind no anatomical changes. Kussmaul, Heubner, Zambaco, Gros and Lancereaux[43] describe cases where the Landry symptom-complex developed during the early stages of syphilis without prodromal symptoms or signs of meningitis. Alexander[44] mentions a case with syphilitic history, where, following ocular palsy and optic atrophy, Landry's paralysis developed and terminated in death.

All these cases were reported years ago; and Nonne[45] observes that in recent literature the views of Heubner as to the existence of a syphilitic form of Landry's paralysis are not confirmed, although he admits that here and there a case may be found with a syphilitic history, as, for example, Goebel's[46], he holds the reverse is the rule. Nonne's contention on this point, may, I think, be allowed, and yet it seems to me that it must be admitted that occasionally syphilis is the etiological factor. In no case I have been able to find does this seem so clear as in the third case I have recorded in this paper. Fr. Schultze, however, reports a fatal case of acute ascending atrophic paralysis[47] with bulbar symptoms in which the onset occurred five months after a syphilitic infection. Schultze himself does not insist that syphilis was the etiologic factor, although other factors were not apparent.

Brissaud[48] records the case of a man aged thirty-three who, following violent pains in the lumbar region, was seized with a rapidly ascending motor paralysis, marked bulbar symptoms and loss of control of the sphincters. Sensation was unaltered. Atrophy did not appear. The knee-jerks were lost but afterwards reappeared. The electric reactions were normal. Slight fever was present. The patient made a rapid recovery.

A case reported by Vizioli[49] most of all resembles my third case. A man aged 65 was infected with syphilis at the age of 21 but not thoroughly treated. The disease began with severe pains followed by motor weakness, first in one hand but afterwards involving the entire musculature, accompanied by pains and fibrillary twitchings and followed by progressive atrophy. Reactions of degeneration were present. The symptoms differed from those seen in the Duchenne-Aran muscular atrophy in that pain was a prominent symptom and that paralysis preceded atrophy.

Nonne[50] reports two cases of progressive anterior poliomyelitis of a descending type. In both the sphincter function was disturbed and paresthesia was present, points which convinced him that the morbid process was not confined to the anterior gray horns. In both cases the disease was arrested after much atrophy had occurred, following a vigorous anti-syphilitic treatment. In both cases obvious signs of syphilis were present.

Summing up now the evidence afforded by this review of the literature and the study of my own cases, it seems that in the symptomatology of Landry's paralysis sensory symptoms are almost constantly present and generally precede the motor paralysis by several hours or a day, occasionally by much longer periods. The most common sensory disturbance is paresthesia, but hyperesthesia, tenderness and spontaneous pain, which may be severe, are not rare. The motor paralysis is usually of an ascending type, but may be of the descending variety or begin in all four extremities simultaneously. As a rule muscular wasting does not occur and electrical reactions of degeneration are absent, but well-marked exceptions to this rule occur. In a few cases fibrillary twitchings are recorded. The sphincters are involved in about one-third of the cases. The four most constant marks of the disease are rapidly ascending motor paralysis, lost knee-jerks, absence of marked fever and preservation of the mental integrity; but occasional exceptions occur. Oppenheim[51] observes that cases corresponding to the original Landry type are easily separated from other clinical types, but that the only absolute criterion for all cases is a flaccid paralysis

affecting in rapid succession the muscles from below upwards or rarely, from above downwards.

But considering the actual clinical records of reported cases, admitting the atypical and "transitional forms," it would seem that we have in Landry's paralysis a symptomatology which stands midway between multiple neuritis on the one hand and poliomyelitis with bulbar paralysis on the other hand, and which at both extremes of its types touches and indeed overlaps the clinical picture of these two affections. Most of the cases of the disease are, however, clearly distinguishable from either multiple neuritis or poliomyelitis. Moreover, the morbid anatomy of Landry's paralysis, like the clinical picture, reveals features common to both multiple neuritis and poliomyelitis; but it reveals changes not common to either, and in some cases carefully examined by the later histologic methods no morbid changes were found.

The various writers on Landry's paralysis are almost a unit in saying that it is due to an infection or toxemia; and this view seems to be founded upon three facts chiefly: (1) the acute onset and rapid course; (2) the presence of bacteria found in a certain number of cases examined post-mortem; (3) the frequent enlargement of the spleen and lymph glands.

In the earlier reported cases collected by Bailey and Ewing a considerable number showed no post-mortem changes. But in the later reports of autopsies examined by the means of the present day exact histologic technique morbid changes of greater or less extent have been pretty generally recorded. Bacteriologic findings have not been so frequent as histologic changes. In some cases the pathologic changes recorded have been so slight as to scarcely afford a reasonable explanation of the symptoms. Oppenheim[53] gives the prudent caution that the finer the technique, the greater should be our caution in accepting the findings revealed by it. But even when examined by the most approved modern methods by competent investigators there are a certain number of cases in which the post-mortem and bacteriologic findings were negative. In a carefully examined case reported by Girandeau and Levi[54] and A. Schultze[55] in 1898 and Kapper[56] in 1900, the post-mor-

tem findings were negative; and in the carefully reported case by Goebel[57] the peripheral nerves, spinal gray matter, anterior and posterior nerve roots were normal; only a light grade of degeneration of pyramidal tracts high up was discovered, insufficient, in the opinion of the writer, to account for the symptoms.

The various post-mortem findings recorded need not at this time he rehearsed since they have been sufficiently analyzed by recent writers. Suffice it to say that they have been most manifold both in degree, situation and kind. Disseminated inflammatory areas in the bulb were found by Ormerod, Boinet, Courmont, while others (Eisenlohr, Schultz, Ketli, Halva and Immerman) have found similar changes in the cord. A great many cases are recorded in which the changes were in the anterior horns of the cord. (Bickel, Taylor and Clark, Worcester, Hirtz and Lesne, Wappenschmidt.) In many others they were in the peripheral nerves (Barth, Dejerine, Bois, Dydynski, Eisenlohr); while in a still larger number both the anterior horns and the peripheral nerves were involved (Centanni, Eisenlohr, Pribykow, Thomas, Knapp and Thomas, Boinet, Mills and Spiller, Krewer, Schultz). In Goebel's case some fresh degeneration of the muscles was detected, although the peripheral nerves and the gray matter of the cord were normal. In a few cases (Diller and Meyer, Goebel, Bickel), changes were found in the white matter of the cord.

A most interesting seat of morbid changes, in view of the constant presence of sensory symptoms, is the posterior nerve roots. Changes in these structures are recorded by Knapp and Thomas, and Boinet. In view of the prominence of sensory symptoms in Landry's paralysis it is much to be regretted that there are very few records of examination of the posterior spinal nerve ganglia. Finally the frequent records of enlargement of the spleen and lymphatic glands and pulmonary hemorrhages must be borne in mind.

The nature of the morbid changes are manifold, representing many forms and degrees of degeneration, extravasation, hemorrhage and inflammation.

While bacteriologic examinations have not so frequently as

histologic examinations yielded positive results, still there are now on record a considerable number of cases in which micro-örganisms have been found post-mortem. The streptococcus, a diplococcus and staphylococcus have been found in the central nervous system. Piccinno found bacteria in the perivascular lymph spaces; Courmont and Bonne a diplococcus in the cerebro-spinal fluid. Remlinger[59] produced acute ascending paralysis in rabbits by injections of the streptococcus and recovered the organisms from the cord.

The etiology is one of the least studied, least understood, and at the same time most important phases of the subject. Bailey and Ewing mention as etiologic factors typhoid fever, syphilis, diphtheria, exposure and over-exertion; while in the great majority of cases analyzed by them (26), there was no definite infection. Various authors state that the disease has followed pneumonia, whooping-cough, small-pox, influenza and septicemia. In several cases alcohol was thought to have been the cause. Oppenheim[58] mentions one case in which the disease followed the kick of a horse who was affected by septicemia. In no case does the etiology seem quite so clear as in one of my own cases recorded in this paper where the disease began a few weeks after the patient had contracted a chancre. In Baumstark's[59] cases malaria was the apparent cause.

There are three views regarding Landry's paralysis at which it may be worth while to glance.

One set of observers have regarded it as nothing more than a form of multiple neuritis. (Dejerine, Barth, Ross, Putnam, Walton, Dydynski, Krewer.) These observers point to the frequently reported changes in the peripheral nerves, and explain the changes occurring in the spinal gray matter on the theory that these are merely parts of the same trophic and anatomic unit, and liken them to the changes which occur in typical multiple neuritis. Against this view may be urged the inconstancy of these morbid changes, the rather frequent cases in which the peripheral nerves exhibited no changes, the fact that the peripheral nerve changes found consist usually of degeneration, seldom of inflammation; and finally that the clinical course of the disease differs considerably from that of true multiple neuritis.

Bernhardt and Westphal concluded that Landry's paralysis is very like acute poliomyelitis, differing from it chiefly because it is more generalized over the gray matter of the cord. Bailey and Ewing include six cases of so-called poliomyelitis in their list of analyzed cases, because of the wide extent of involvement and the presence of bulbar involvement. This view does not sufficiently account for the ascending character of the disease or the frequent sensory symptoms. Cases followed by complete recovery also afford an argument against it.

The third and most recent view of Landry's paralysis, is that supported by Thomas and Knapp, and Thomas, and Mills and Spiller: that it is primarily an affection of the motor neurone of the first order, a parenchymatous degeneration of toxic or infectious origin. There is much to support this view of the morbid anatomy of the disease in the consideration that the most constant pathologic changes are those found in the anterior horns of the cord, and that such a theory offers a reasonable explanation of the clinical symptoms recorded in many of the cases. But arguing against this view is the post-mortem record made by Knapp and Thomas themselves, of degeneration in the posterior nerve roots, a similar record made by Boinet, the case of Schwab where the only change was great vascular congestion, the cord and peripheral nerves being normal, and the cases with negative post-mortem findings.

To these three views might be added the view maintained by Taylor and Clark[60] and supported by Schwab, that there is no such "disease" as Landry's paralysis; that the cases described as such exhibit "no essential constancy in the clinical symptoms nor in the pathologic findings, and the etiology is wholly vague."

While it is quite true that the symptomatology, morbid anatomy and etiology of Landry's paralysis are inconstant and variable, and that it is not a disease in the sense in which tetanus or small-pox or exophthalmic goiter are diseases, it is at least as much of a "disease" as some other so-called "diseases," *e.g.*, cerebral palsy of childhood.

The term Landry's paralysis cannot be dropped. It has been used too long to be erased from our nomenclature. Nor

is it, in my opinion, desirable that it should be dropped; but it is most desirable that the older conception of its meaning be enlarged to include this highly striking group of cases, variable though they be, standing both in symptomatology and pathology midway between multiple neuritis and poliomyelitis, and which cannot be placed under either of these headings. It must be borne in mind that these cases exist, and if the name of Landry's paralysis were discarded a name would have to be invented under which to include them. No harm can come of the continued use of the term Landry's paralysis if the variability in the etiology, symptomatology and morbid changes and bacteriologic findings be borne in mind, and if we at the same time freely admit the contention of Taylor and Clark, that in the strict sense of the word it is not a "disease" but rather a group of symptoms, overlapping each other more or less, and which are toxic infections in origin, produced by a great variety of poisonous agents.

The accumulated evidence points very strongly to the toxic origin of Landry's paralysis; and with equal certainty it indicates that it is produced by no one single agent but by many. Multiple neuritis may be here briefly glanced at. This disease is produced by traumata, toxic agents, and slow degeneration changes. Among the toxic agents may be mentioned lead, arsenic, copper, carbon monoxid, the toxines of diphtheria, influenza, malaria, typhoid fever, rheumatism, etc. The symptomatology of multiple neuritis varies greatly, depending upon the nature and potency of the toxic agent, and the resistance or vulnerability of the tissues. The poisons of lead and diphtheria possess peculiar selective action. Similarly the symptomatology of cerebro-spinal meningitis varies enormously; and the disease is doubtless susceptible of being produced by several varieties of toxines, perhaps by many. Putnam[61] has well said: "When the nervous system is working in a perfectly normal manner and not under strain it seems physiologically a unit. When the reverse is true it becomes evident it is comprised of many parts of varying powers of resistance to disease."

For a definition of Landry's paralysis I would suggest the

following: Landry's paralysis is an infection or toxemia produced by various poisons in which the spinal cord (including the bulb) and the peripheral nerves, and especially the lower motor neurone, are attacked, and in which parenchymatous degeneration, hemorrhagic and inflammatory exudates of various kinds and degree, are usually found post-mortem in this portion of the nervous system. But in a few cases no post-mortem changes are discoverable, and in still others changes which are inadequate to explain the symptoms. The spleen and lymph glands are usually enlarged. Bacteria of various kinds have, in a considerable number of cases, been found in the central nervous system; but in the great majority of cases a careful examination has failed to discover any microörganisms. The male sex is affected much more commonly than the female. No age is exempt.

The first symptom of the disease is usually paresthesia, sometimes accompanied by pain in the members which are subsequently paralyzed, and this may exist from a few hours to several days or even longer as a premonitory symptom. General malaise, neurasthenic symptoms and ataxic movements may also be premonitory symptoms. Widespread flaccid paralysis rapidly develops involving legs, trunk and arms, and, in fatal cases, the muscles innervated from the bulb. The paralysis is usually of an ascending type involving the legs first, but it is occasionally of a descending type and may involve all extremities simultaneously or attack the various muscles in an irregular order. In the majority of cases the sphincters are intact. Their involvement however is not rare. The knee-jerks are absent. As a rule atrophy, fibrilliary twitchings and electrical changes in the muscles are absent; but any or all of these phenomena may be present. Pain and tenderness to pressure may be prominent symptoms, but as a rule are not very marked, although they are present in some degree in the majority of cases. As a rule the temperature is normal or but slightly elevated. Consciousness is almost invariably preserved unimpaired throughout the attack.

Cases which terminate fatally run a course of from a few days to one or two weeks, and almost always exhibit bulbar

symptoms. In cases which do not terminate fatally the course may extend over many weeks or even months, and recovery may be incomplete. Often premonitory symptoms exist for several days or even weeks.

While the prognosis is grave, a considerable number of cases terminate in recovery or partial recovery.

The treatment should be directed to the accomplishment of two ends: the support and stimulation of the vitality and the production of elimination. To this end hypodermic injections of strychnia and the use of whiskey, digitalis, mild diuretics and cathartics are to be recommended. Anodynes may be required when pain is severe. In cases in which syphilis has been recently contracted, vigorous specific treatment should at once be instituted. Later, after a turn for the better has occurred, massage, electricity and hydrotherapy may aid the tendency to recovery.

Note.—Since the above was written an important article on "Poliomyelitis in the Adult," by Dr. E. W. Taylor, has appeared in the August (1902) number of this journal, in which the clinical histories of six cases are given. In Taylor's first case in which the clinical manifestations pretty closely corresponded to the Landry symptom-complex, a careful post-mortem examination showed evidence of primary inflammation of the ventral horns of the cord, and secondary destruction of the nerve cells, and marked degeneration of the peripheral nerves.

Taylor reiterates his objection to the term Landry's paralysis and observes that "Poliomyelitis is none the less poliomyelitis because it simulates in its clinical course what Landry, many years ago, described as acute ascending spinal paralysis." This contention may be readily allowed. But all cases of so-called Landry's paralysis can not be shown to be cases of poliomyelitis; many more closely correspond to multiple neuritis. I can see in Taylor's argument no reason for changing the views I have expressed in this paper.

I would suggest that the term Landry's paralysis be employed precisely as most neurologists employ the term apoplexy. Under this term is included both cerebral embolism and hemorrhage, and also those rarer and more or less obscure conditions

which may produce apoplectic symptoms. When we feel sure that an apoplexy is hemorrhagic in character, we speak of cerebral hemorrhage; but since we see many cases in which the differential diagnosis between cerebral hemorrhage and embolism is uncertain, the more general term apoplexy becomes convenient.

Similarly the term Landry's paralysis may be conveniently retained to designate cases of a certain clinical type, some of which can be made out to be cases of poliomyelitis, others of multiple neuritis. But the term, like apoplexy, is especially useful to designate those cases which cannot be made out to be either cases of multiple neuritis or poliomyelitis and those cases in which the post-mortem findings are negative.

With advancing knowledge the terms apoplexy and Landry's paralysis may be discarded, but that day is not yet.

[2]Amer. Jour. Med. Sc., August, 1898.
[3]Jr. Nerv. and Ment. Dis., 1898, p. 369.
[4]Amer. Jr. Med Sc., April, 1896.
[5]N. Y. Med. Jr., July 4-11, 1896.
[6]Jr. Nerv. and Ment. Dis., June, 1898.
[7]Amer. Jr. Med. Sc., Aug., 1898.
[8]Jr. Nerv. and Ment. Dis., Feb., 1900.
[9]*Ibid.*, April, 1900.
[10]"Leçons sur les maladies du systéme nerveux," Deuxieme Serie.
[11]"Lehrbuch der Nervenkrankheiten," Zweite Auflage p. 397, et seq.
[12]Jr. Nerv. and Ment. Dis., Dec., 1900.
[13]*Op. Cit.* p. 192.
[14]*Op. Cit.*
[15]"Semiologie du système nerveux," p. 559.
[16]"Vorlesungen über die path. Anat. des Rückenmarks," 1901, p. 295.
[17]Neurolog. Centralblatt, April 1, 1898.
[18]*Op. Cit.*
[19]*Op. Cit.*
[20]Quoted by Knapp and Thomas, *op. cit.*
[21]Med. Rec. Vol. XLVII., p. 534.
[22] *Op. Cit.*
[23]Jr. Nerv. and Ment. Dis., May, 1898.
[24]Bull et Mémoires de la Sociète Méd. des Hôpitaux de Paris, 1895, p. 659.
[25]La Presse Médicale, 5, 1897, p. 269.
[26]Abstracted in Jr. Nerv. and Ment. Dis., Vol. 26, p. 239.
[27]Bull. et Mémoires de la Sociète Méd. des. Hôpitaux de Paris, 1895, p. 63.
[28]Brit. Med. Jr. I, 1898, p. 1,654.
[29]Jr. Nerv. and Ment. Dis., 1901.
[30]Obozrenjepish, 1899, No. 2, p. 151 (Abstracted in Jr. Nerv. and Ment. Dis.).
[31]Brit. Med. Jr., May 4, 1901.

[32]Jr. Nerv. and Ment. Dis., Dec., 1900.
[33]Deutsche Zeitschr. f. Nervenhk. Bd. 16, H 3-4, p. 305.
[34]Lancet, 1900, Vol. II., p. 1490.
[35]Wiener klin. Wochenschr., 1900, No. 1.
[36]Jahr. f. Kinderhk. Bd. 51, H. I., p. 67.
[37]Amer. Jr. Med. Sc. Vol. 120, p. 36.
[38]St. Bartholomew Hospital reports. Vol. 36, 1900, p. 137.
[39]Inaug. Dissert. München.
[40]Jahresbericht über Neurolog. Psych. 1900, p. 406.
[41]Berlin klin. Woch. 1900. No. 37 and 38.
[42]"Syphilis und Nervensystem," p. 297.
[43]Quoted by Nonne. *Op. Cit.*
[44]"Syphilis und Auge." Wiesbaden, 1889. Quoted by Nonne.
[45]*Op. Cit.*
[46]*Op. Cit.*
[47]Berlin klin. Wochenschr., 1883. No. 39. Quoted by Nonne.
[48]Rev. Neurologique. Apr. 30, 1902, p. 355.
[49]Ann. di. nevrol. Abstracted in Neurolog. Centralbl., 1900, p. 79.
[50]*Op. Cit.*
[51]"Lehrbuch der Nervenkrankheiten," p. 401.
[52]"Lehrbuch der Nervenkrankheiten," p. 400.
[54]Rev. Neurologique, Oct. 1898.
[55]*Op. Cit.*
[56]Wien. klin. Wochenschr. 1900, No. 7.
[57]*Op. Cit.*
[52]La Semaine Méd., July 15, 1897.
[58]*Op. Cit.*
[59]Berl. klin. Woch., 1900. No. 37 and 38.
[60]*Op. Cit.*
[61]*Op. Cit.*, p. 230.

NOTE ON CELL CHANGES IN A CASE OF COMPLETE COMPRESSION OF THE CORD.[1]

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The mode of connection between neurones, whether motor or sensory, is still one of the unsettled questions of the finer anatomy of the nervous system. In intimate relation to this question is that of the effect of destruction of one neurone upon the nutrition and structure of those which are in connection with it. The case presented today seemed to me peculiarly fitted for the study of changes in the peripheral motor neurone when the axis cylinder of the central neurone has been completely destroyed, and this both because the destruction of the motor neurone of the second order was unusually complete, and also because of the long duration of the symptoms, for a period of thirteen years.

The case was presented to this Association at its meeting in Washington two years ago[2], and a brief résumé of the clinical features of the case will suffice.

The case was one of endothelioma of the dura. The patient, a single woman, at the age of twenty-one, in June, 1884, began to lose the use of the legs. In October she could still walk with assistance, but in April, 1885, she had no power in the legs, though the arms were unaffected. There was incontinence of urine at this time. In September, 1887, it was noted that there were violent, spasmodic and sudden contractions in the limbs. In November of that year there were noted flexor contractures in the legs. In August, 1892, it was noted in the records that there was no sensation in the legs, and no perception of pain. The cranial nerves were not affected, and although the pupils were

[1]Read at the annual meeting of the American Neurological Association, June 5, 6 and 7, 1902.

[2]Thomas, J. J., "Two Cases of Tumor of the Spinal Cord." Journal of Nervous and Mental Disease, November, 1900.

pretty constantly dilated they reacted to light and upon accommodating. The upper extremities were not affected, while the lower ones showed extreme contractures and were atrophied to a marked degree. The reflexes were not obtainable on account of the contractures. Sensibility in all forms was lost to a level on the trunk at about the mid-dorsal region. The function of the vesical and rectal sphincters was completely abolished. Decubitus was present. Exitus from exhaustion occurred on April 19, 1898. Autopsy by Dr. R. M. Pearce on April 19, 1898. The decubitus and contractures of the lower limbs were noted. In the mid-dorsal region of the vertebral column there was a projecting knuckle formed by the spinous processes of two vertebræ. Upon removing the cord there was found below the cervical enlargement and at the level of the spine of the scapula a granular, reddish swelling of the dura, about 2.5 cm. in length. This tumor appeared to be meningeal, but had almost completely replaced the cord. Below this point the cord appeared considerably atrophied. There was no lesion of the cord opposite the vertebral knuckle. Besides the conditions just spoken of, there was found diffuse nephritis, and cystitis. The microscopical examination of the tumor showed it to be an endothelioma of the dura, with the peculiar concentric rings seen in the tumors called psammomata. This tumor had produced what was practically a complete compression of the cord in the mid-dorsal region, the only axis cylinders which retained their myelin sheaths being a very few scattered ones at the periphery of the cord, and a few at the periphery in the posterior columns. There was a marked increase of the neuroglia in this part of the cord. Above the point of compression the cord showed marked degeneration of the median part of the posterior columns, and of the direct cerebellar tracts, and a more diffuse degeneration of the periphery of the cord throughout the lateral border and along the anterior fissure. Below the point of compression the cord was smaller than normal, and there was a well marked indentation just anterior to the point of exit of the posterior nerve roots. There was a very marked degeneration of the lateral and anterior pyramidal tracts, and a moderate diffuse degeneration at the periphery of the cord in its anterior and lateral

PLATE I.

PLATE III.

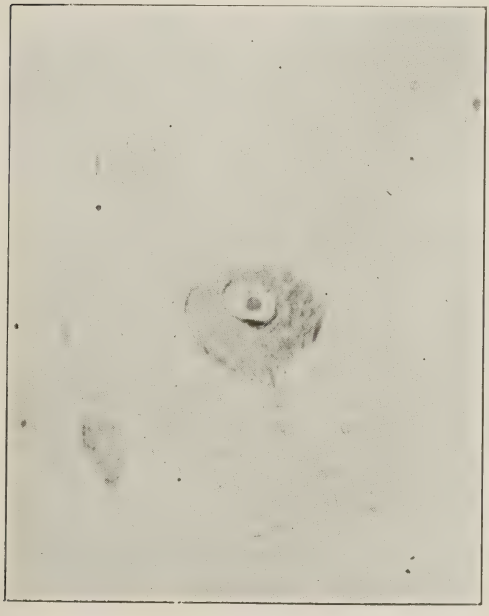

PLATE II.

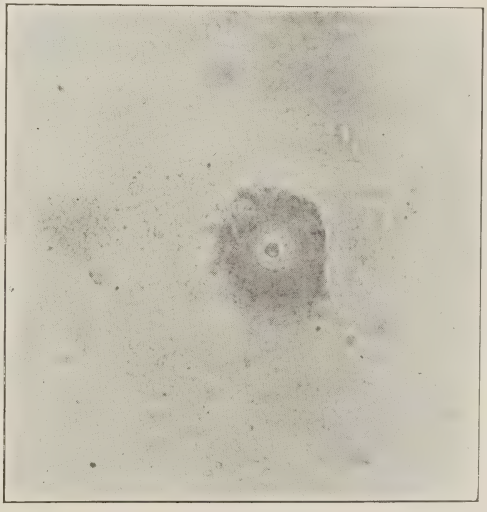

PLATE IV.

DESCRIPTION OF PLATES:

Plate 1—Cells from the ventral horn of the lumbar enlargement. Magnification about 250 diameters. Nissl's stain. Normal cell.

Plate 2—Do. Cell showing almost complete destruction of the chromophilic granules. Irregularity of nuclear membrane.

Plate 3—Do. Cell showing diffuse staining of the cytoplasm. Poorly staining nuclear membrane.

Plate 4—Do. Cell with diffuse staining of the cytoplasm and advanced nuclear changes. Vacuolization of nucleolus.

parts, and a very slight diffuse degeneration throughout the anterior ground bundle. The degeneration at the periphery of the cord was no longer seen at the lower part of the thoracic cord, though the degeneration of the motor tracts, both lateral and anterior, was well marked here, as also that of the lateral pyramidal tract in the lumbar cord after the anterior tract had ended.

Since writing the previous report of this case a portion of the lumbar enlargement of the cord, which was hardened in formalin, was embedded in paraffine and stained with methylene blue by Nissl's method. The large motor cells of the ventral horn of the lumbar enlargement show occasional cells in which the chromophilic granules appear normal. These are irregular in shape, with distinct outlines, leaving the axonal hillock free, and they can be traced into the protoplasmic processes of the cell for fully the normal distance. The nucleus is clear and the nuclear membrane distinct. In some of the cells the nucleolus appears slightly swollen, and stains irregularly, and takes a slightly purplish tinge with the methylene blue stain. These normal cells are on the average two or three in each section of the cord. The remaining cells of the ventral horns show well marked changes. In most of them the granules at the periphery of the cell appear normal, and can be traced as usual well into the dendritic processes. The cell processes appear normal also. About the nucleus and filling the whole central part of the cell cytoplasm are very fine granules, in many cases so fine as to give merely a faint bluish tinge to the protoplasm about the nucleus. The axonal hillock can usually not be seen distinctly and seems to contain the same fine granular dust. The nuclear membrane is either very indistinct, or, as is more usually the case, the outline of this membrane is extremely irregular, so that in places it cannot be seen at all, and at other parts of the periphery there is a heaping up of chromatin substance which makes an irregular mass at one or two points of the border of the nucleus. The body of the nucleus is as a rule quite clear. The nucleolus is as a rule larger than normal, and generally vacuolated, and has a distinct purplish tinge. The nucleus, as a rule, occupies the normal position in

the center of the cell-body, but occasionally it is placed slightly excentrically, and in a good many instances the nucleolus is placed much nearer the periphery of the nucleus than is normally the case. In many of these cells is seen an area, sometimes a quarter or a third of the area of the cell, which is clear from granules or granular dust, and is of a yellowish color. This is apparently caused by an increase of the pigment granules of the cell, though the granules themselves are not distinctly seen even with a high power immersion lens. The few small nerve-cells at the base of the dorsal horn at this level of the cord appear practically normal.

We have in this case, to summarize, a tumor of the dura which produced a complete compression of the cord, resulting in a complete paraplegia with the formation of marked contractures and an entire loss of all forms of sensation, and loss of the control of the bladder and rectum, which condition lasted for thirteen years. There was a general smallness of the cord below the point of compression, and the usual systemic degeneration of the pyramidal tracts. The large motor cells of the ventral horn of gray matter in the lumbar enlargement were in a considerable number of cases found to show no changes in the chromatic substance as studied by Nissl's staining method. The larger number of these cells, on the other hand, showed well marked changes, consisting in a central chromatolysis, together with marked nuclear changes, as shown by the disappearance of the nuclear membrane and the heaping up of the chromatin substance at one point at the border of the nucleus, as well as by the slighter changes occasionally found where the nucleus took a diffuse stain with the methylene blue, or where there was swelling and irregular staining of the nucleolus.

It is difficult to imagine a case more suited than this to the elucidation of the question of possible changes in the peripheral neurone secondary to destruction of the cerebral neurone, the so-called reaction at a distance, both because of the long duration of the condition, and the completeness of the interruption of conduction through the cord of all impulses, as shown both by the symptoms during life, and by the results of the microscopical examination of the cord. The conclusions which seem

to be justified may appear somewhat contradictory, but they certainly seem to be that a central chromatolysis and nuclear changes of quite a severe grade may occur in nerve cells of the ventral horn when interruption of conduction from the brain of motor impulses has been cut off for a long time; and second, that in spite of this interruption of conduction from the central neurones the peripheral motor nerve cell may retain its normal cytoplastic appearance for many years.

Perhaps the most obvious inference from these facts is that recovery from the symptoms produced by a compression of the cord from without, as from a tumor of the dura as in this case, is probably possible as far as the preservation of the peripheral neurones is concerned long after the entire loss of any hope of recovery of the central neurones after removal of the compression.

A CASE OF SEVERE HYSTERICAL CONTRACTURE OF THE LEG, AND ITS TREATMENT.[1]

By Frank R. Fry, A.M., M.D.,

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The special object of this communication is to recount the difficulties encountered in the treatment of a serious hysterical contracture. I shall therefore be as brief as possible, only dwelling on various features of the case sufficiently to reveal the nature of it.

The contracture developed in the spring of 1900, the girl being then 13 years old. The following is the best account I could obtain of it: In January, 1900, she had a severe cold, attended with much aching in all portions of the body. After a few days she complained especially of the whole left lower extremity, and refused to put the heel to the floor in walking. Soon after her mother noticed that it was beginning to draw up, or contract. The physician then in charge tried to straighten it by bandaging it to a splint. This always aggravated matters. From the mother's description it is evident that the first jerking and hysterical attacks began in these attempts at bandaging the leg. The physician directed the mother to apply the splint daily. This became every day increasingly difficult. Finally one day, during the process, the heel flew up to the buttock with great force. The splint was hurled across the room by the force of the contraction; and the leg remained in this extreme flexed condition. The seance terminated in a full-fledged hysterical paroxysm. This occurred two months after the patient first began to complain of the leg. After this she had a protracted attack almost every night. The mother relates that for three months the nights were full of returning terrors to herself and the child on account of these attacks. She remained in bed about eight months. The jerking in the leg meantime began to gradually subside and the attacks became less severe and less frequent.

I first saw the patient March 19, 1901 (the contracture being then one year old). Her mother, from whom I obtained the major portion of the history, accompanied her to the city. The parents were becoming very anxious about the leg. After

[1]Read by title at the Annual Meeting of the American Neurological Association, June 5, 6 and 7, 1902.

consulting the leading regular practitioners in their vicinity, they had employed various irregulars, including "osteopaths." From the latter especially they had hoped for much. But nothing had seemed to make any impression on the leg. Although

Hysterical contracture. (A similar contracture is figured in Oppenheim's "Diseases of the Nervous System," American edition, p. 680.)

the general condition had improved considerably, the mother was very confident that there had never been the slightest relaxation of the contracture during sleep or under any other circumstance.

Dr. Philip Hoffman, who kindly looked after the orthopedic side of the case, describes below the condition. His description and the accompanying photograph reveal at once the character and intensity of it. I may add a few words, however, to complete

the history. She was placed in a hospital and carefully taken care of and observed. The first and second nights after her arrival the nurses were called to her room to find her in a frightened state, as they called it, the condition being very similar to ordinary night-terrors in a child. The mother assured us that this was the slight remaining remnant of the former severe hysterical attacks, and that it came every night soon after falling to sleep, but that of late it soon passed and the patient usually rested the remainder of the night. A hypodermic injection of 1-100 gr. of hydrobromate of hyoscine was given at bed-time. After the first or second dose the spells disappeared and have not recurred since. The drug was soon discontinued and blank injections kept up nightly for some time.

In the hospital we soon found that the patient was a bright, intelligent girl. She had a pleasant disposition, and readily adapted herself to the new surroundings. She never made complaints, and seldom alluded to her disability in any way. When the limb was more rudely handled, her objections to the manipulations were usually manifested only by silent resistance and never by any great outcry. I soon found that she was very anxious to have the deformity corrected, but she did not seem very hopeful about it. She used one crutch, and got about with it in lively fashion. Her general condition improved rapidly, due less to the medicine and other therapeutic measures than to the fact that with the spring season she could be out in the air a great deal.

My physical examinations were made casually and in piecemeal manner, and kept free from suggestive features. I found it difficult to determine the sensory conditions of the skin in the various portions of the limb. The hyperesthesia and alertness of the muscles were so intense that during an examination a stimulus of almost any kind threw them on their guard. The consequent spasm was evidently very painful, producing a state of anxiety and mental confusion which could not be assuaged by any assurance, and which made the sensory tests always unsatisfactory. The thigh seemed always to be quite hyperesthetic to all kinds of stimuli up to two inches above the groin. The leg and foot were generally somewhat, but never totally anesthetic, and more so on the anterior aspect.

There was pronounced pharyngeal anesthesia and also narrowing of the visual fields. Both of these conditions were much relieved with her general improvement. Aside from those mentioned, no hyperesthetic, anesthetic, or hysterogenic regions were found.

She was given tonics and a careful hygienic regime, including brush and salt rubs, cold ablutions to the spine, electricity, etc. She was never hypnotized, but many milder suggestive meas-

ures were tried. Her general condition improved until she seemed to be in perfect health and spirits. There was no change, however, in the condition of the contractured member.

On the 7th of June (after she had been under observation for two and a half months) I introduced into the spinal theca a little over half a grain of tropa-cocain. I used the tropa-cocain, and only a small dose, instead of the hydrochlorate of cocain, because I wished to have the constitutional symptoms as slight as possible. There was some flushing of the face and fulness in the head which was hardly complained of, and lasted for half to three-quarters of an hour. The anesthesia was not complete, the result of the injection being quite disappointing. However, there was some relaxation of the muscles, so that the heel was separated from the buttocks three to four inches for several hours. When the effect of the cocain had passed the contracture was as rigid as ever.

The especial reason for using spinal anesthesia and at the same time producing as little general disturbance as possible, was the hope of a suggestive effect which might enable us to introduce some process of gradual extension. There was so little promise in the procedure, however, that before repeating it I determined to produce complete chloroform anesthesia and discover the condition of the tendons and ligaments of the knee joint. This was so much worse than what I had hoped to find that at this juncture I called Dr. Hoffman to my assistance. He agreed with me that it was better, all things considered, to forcibly extend the leg at once. In deciding upon this action instead of first trying some more gradual process of extension, we were influenced especially by the following considerations: First, the decided, hysterical resentment toward restraining apparatus, the use of which would be necessary in a gradual process; and to this was added the extreme muscular hyperesthesia, which in all probability would only be controlled by total anesthesia. Secondly, careful measurements showed that the leg was considerably shorter than its fellow. The girl was growing rapidly, the disparity in the length of the extremities probably just as rapidly assuming a more important proportion.

The following is Dr. Hoffman's account of the condition and of the operation which he performed for its relief:

"On examination, June 17, 1901, the right knee was found in a condition of such extreme flexion as to cause the heel to indent the buttock. The hamstrings, especially the inner, were in a condition of tonic spasm and somewhat tender on pressure. The power of voluntary extension was entirely absent. The contractions became more marked, and the patient complained of pain whenever the slightest attempt was made to passively ex-

tend the leg. The right lower extremity was atrophied. Measurements showed that the right tibia was twelve millimeters, and the right femur ten millimeters, shorter than the left; a total of twenty-two millimeters for the whole limb. The circumference of the right calf was forty-four millimeters, and of the right thigh, at its middle, forty-one millimeters less than that of the left. The right foot was nine millimeters shorter, and its circumference over the ball twelve millimeters, and over the heel six millimeters less than the left.

"On June 26, while the patient was under the influence of chloroform, it was found that with considerable force the limb could be straightened only sixty-five or seventy-five degrees, that is, to not quite a right angle. Accordingly, under aseptic precautions, all the hamstrings were subcutaneously and thoroughly divided about three centimeters above their insertions into the tibia and fibula. The limb could now be readily extended to well beyond a right angle, but great resistance was again encountered when the leg was brought to about sixty degrees from a straight line. This was apparently due to shortening of the posterior ligament. The skin was also very tense. To secure better leverage and to prevent the head of the tibia from slipping into the popliteal space, a genuclast was applied, and, after considerable effort, the limb was brought into a straight position. This left a gap of about twelve centimeters between the ends of the cut tendons. During the latter part of the operation the stretched skin tore and retracted, leaving a denuded surface about eight by five centimeters in area. The tear, fortunately, did not take place over the tendon wounds, but about five centimeters below. The wounds were dressed and the limb retained in the straight position by means of a plaster of Paris dressing extending from the toes to the perineum.

"In eight weeks the separated proximal and distal portions of the cut tendons had become firmly united by new material growing into the gaps. On September 26, there being no tendency of the hamstrings to again contract, the plaster of Paris splint was discarded and a light steel brace, allowing motion at the knee, applied. Four months after the operation the patient could walk with fair use of the hamstrings. A thick sole was attached to the shoe of the affected limb to compensate for the shortening of twenty-two millimeters, which, of course, was not lessened by the operation."

The patient left the hospital for home December 14, almost six months after the operation. At that time there was a complete reaction of degeneration in all the muscle groups of the leg and foot, and a total loss of sensation of all kinds below the knee. For two months she had been having almost daily an electric massage of the foot, leg and thigh with a strong gal-

vanic current. The temperature and color of the affected member were remarkably good considering the extent and degree of the paralysis.

The following is a letter received May 7. It conveys the latest account I have of the patient, and at the same time a good idea of her mental capacity (above that of the average girl of fifteen years).

"Dear Dr. Fry:—After this long silence I am writing you my troubles again. My foot is entirely well and I have been wearing my brace quite a while. I also take the electric treatment all the time. Since spring came I have felt so much stronger and better in every way until the last two weeks. My head aches almost constantly. Have indigestion and feel so bad when I first awaken in the morning. But after I'm up awhile it wears off to some extent. I believe I am somewhat run down, that is, my general health not as satisfactory as it might be. I've been almost living out of doors during this lovely weather, walking, driving and riding horseback, too, some.

"If you have any suggestions to make concerning my condition, would be glad to have you write me at an early date.

"Most sincerely, ————————."

In explanation of her statement, "my foot is entirely well," etc., it may be interesting to note that soon after her return home the great toe of the lame foot was crushed, and at about the same time several large blebs appeared on other portions of the foot and leg. In correspondence with the patient and her mother they could not explain how these traumata were produced. The mother thought that the blisters might have come from the hot-water bag used to warm the foot, but she was not certain about it and seemed entirely mystified over the injury to the toe. I naturally suspected hysterical mutilation, and communicated my suspicion to the attending physician, Dr. S. Sanford; but he was unable to throw any additional light upon the matter. He stated, however, that on account of the bad trophic conditions the repair of the lesions was very slow, and that he was using an elastic bandage with evident advantage. As no more lesions have appeared my suspicions were probably unfounded.

At this writing I am unable to report exactly the condition of the leg and foot. I had expected to see the patient in April or May for an examination. I have had occasional communications from Dr. Sanford, the mother and the patient. They convey no information of importance beyond that contained in the little letter here given. Considering, however, the state of affairs when I last saw her, six months after the operation, it is not probable that the innervation will be greatly improved.

PSEUDO-EPILEPSIES, AND THE RELIEF OF SOME FORMS BY THYROID.[1]

By William Browning, M.D.,
of Brooklyn, N. Y.

It may be well to indicate what is intended by the term pseudo-epilepsy. It is here used in a somewhat provisional sense, to specify a class of cases that can not as conveniently be brought under any other head.

We all know of the ordinary, the Jacksonian or symptomatic, reflex, senile, and such forms; but besides these we meet with occasional irregular cases which, while presenting features suggestive of the genuine type, yet do not prove to be definitely of that character. While these appear to represent a great variety, a careful study of them may simplify the matter and perhaps lead to useful conclusions. The individual case looks plain enough when worked out, but each is a problem at the start, and we need fuller sources of information.

The term epileptiform does not sufficiently distinguish them, nor does it really apply. That refers to the type of separate seizure, rather than to a more or less continuous condition, and, for that matter, some of these cases hardly show epileptiform seizures. Others terms, as epilepsia larvata, minor, eclampsia infantum, etc., are either insufficient or do not include the cases here intended. As it is not certain that they are cases of incipient epilepsy, that term is ruled out.

Another and practical reason for the designation pseudo-epilepsy, is that such cases come to us either already labeled epilepsy, or for us to determine whether they are true epilepsy or not. Quite in harmony with this is the fact that many of these cases are overlooked or ignored, with lasting damage, possibly, in consequence. If we simply speak of the rachitic or other form of trouble, it will fail to catch the ear of those who must first take the initiative. There is therefore practical as well as theoretic convenience in distinguishing such a class.

[1]Read at the annual meeting of the American Neurological Association, June 5, 6 and 7, 1902.

The diagnosis of epilepsy is supposed to be easy, and in many cases it certainly is. Especially since the attention of the profession was directed by Seguin to the symptoms in incipient cases, there is a greater inclination to class all light and not well-defined forms under the main head. In this respect we may readily go too far.

I will give in detail a few cases in which the matter seems to be clear. All are from private practice. The cases to be cited have this in common; they can all be attributed to disturbance or abnormality of nutrition.

Case I—A girl of 5½ years, seen November 7, 1901, with Dr. Childs. Father an Italian, and faints easily. Mother of German race, though born here. This is the oldest child, and of dark complexion, like the father.

The present trouble first appeared on a warm day in the summer of 1899, when she was about 3¼ years old. Soon after they began, *i.e.*, in the summer of 1899, she was 3 weeks in the country, and there was free of attacks. The summer of 1900 she was also free while at the seashore. In 1901 she was in the country from April 13, and remained free up to September 1, when she had one; and last winter even at home she was for some time free.

For the past two months, *i.e.*, since coming home, she has had one attack about every other week. Never has one while in bed, but only when on her feet. The attack may come at any time, morning, afternoon, or evening, on the street or in the house.

She acts about the same way in every attack. "Her face gets like a lobster," it is so red. She seems to get hot all over, and very soon begins to perspire generally (notably trunk, lips and nose). The first thing in an attack is that she shouts, "I'm dizzy." She says her "head goes round and round." The whole thing lasts not more than five minutes, and then she is fully over it. Lying down gives the quickest and most relief. Usually no twitching accompanies the attack, though there was some during a recent seizure. She understands when spoken to, even in an attack, and can control herself to answer. She gets up immediately after an attack and plays as usual. Never vomits after an attack. Excitement has no recognizable influence. The attacks are no severer nor in any way different now from those at first, only more frequent.

The above gives about all there is to say of the seizures themselves. But a number of details may be added regarding her condition in general. She has not as yet been to school.

Dislikes riding in a closed car, though not in an open one. Has been a bashful and socially backward child. At the same time, as the mother says, "she is very cute at getting on to things." No visual, ocular nor pupillary abnormality. Mitral and aortic sounds all clear. Pulse 98, regular and of fair force. She still has a little lisp, or persistence of "baby-talk;" says pin instead of spin, and seep for sleep. No nightmares nor nocturnal frights. Sleeps on her back with head low and mouth shut. Always a long sleeper, 11 hours or more. Has an exceptionally thick head of hair, two long braids. Does not tire easily. The throat looks healthy, and there is a fair pharyngeal reflex. She had pain for a little time in one ear last winter, but the drums now look normal. Somewhat stubborn and headstrong, but not more than other children. No difference in the attacks whether the stomach is full or empty. Inclines to constipation, but that is regulated when necessary. "Is quite a little meat-eater," says the mother. Is plump, but not specially stouter than formerly. No beading of ribs. Spleenic dulness 7½ cm. long. Never any chilliness or rigor, though she flushes readily in a warm room. Temperature, 99.1 deg. A record of her temperature showed a customary, though not invariable evening rise to 100 deg. She was put on small doses of quinine, and went a week without rise of temperature or attack. The quinine was then dropped, when she again showed irregular temperatures, at times as high as 103 deg., always late in the day. Quinine then failed to affect either the fever or attacks, it was stated. At any rate, the seizures were about one every two weeks in January. An examination of the blood at this time showed no malarial organisms.

A more careful hunt for sources of digestive trouble was next made. It was finally discovered that the child was in the habit of every now and then going to her uncle's meat-market and helping herself ravenously to raw meat. Her peculiar craving for this was noted above. It was easy to stop this practice. She has remained quite free from any disturbance the 4½ months since.

Although the cause appears to have been found, there may still be question where to class this case. The frequent rise in temperature negatives any suspicion of reflex. The afternoon character of the febrile rise and the large spleen speak against simple attacks of indigestion. Anything characteristic of rickets was wanting. The case can, I think, most conveniently be put down as one of pseudo-epilepsy due to disturbed metabolism.

Case II—Dolly G., 3½ years old, when first seen in July, 1900, at the request of Dr. McNaughton. She had been under

the care of another physician who considered her disease epilepsy, and had treated her therefor.

No instruments were used at the child's birth. It was a natural, easy delivery, though it was then noticed that she had a large head. The mother has had no miscarriage. There is one younger child, a healthy boy of 17 months. There was a decidedly neurotic heredity. The paternal grandfather took to gambling after middle life, and has since indulged in it whenever possessed of the means. The father is an asthmatic with nasal trouble. A sister of the father is an all-round neurotic of the extreme kind. While the mother is healthy, two half-cousins on that side have suffered from chorea, one long and very severely. In fact the heredity was so bad as to be considered strongly corroborative of epilepsy by one of the physicians who had seen her.

Dolly was a bottle-fed baby, on condensed milk. She nearly died of summer-complaint during the first two years. Otherwise always of constipated habit, with occasionally no movement for three days, but of late this has been regulated. Two years ago in the country, and again last summer, "she was almost one big sore, from mosquito bites and scratching them."

Otherwise she has always been a perfectly bright, healthy child up to two months ago. Her present trouble seemed to start from the time her first vaccination took. The disturbance is rather more at night. She twitches in sleep, rubs her legs together, and falls out of bed. At first this was so bad that convulsions were feared, but chloral and bromide have some control. She sleeps at first quietly up to midnight, but is restless after that. These peculiar nocturnal spells occur nightly if nothing is taken to stop them.

During the same period of two months she has also had so-called "peevish spells" by day. In these she harps on some one thing she happens to desire. Though not cross, she cries and keeps repeating the same thing—hysterical rather than cranky, the mother expresses it. The mother also states that she is "changed in talking," and has now become "childish." A considerable degree of dulness or stupidity is manifest. She is strikingly sober, and is said to have always been of a most serious temperament. Her voice and the questions that she asks are peculiarly infantile, and this is the more noticeable as she is large and stout for her age. She has a plump, wide face, and flattened-out, pug nose. The upper front teeth are gone, except the roots. The neck about the thyroid is small, and no definite gland can be made out. The skin is noticeably coarse and the features heavy. The head is large (over 50 cm.). This combination produces an effect immediately suggestive of myxe-

dema. It has always been noticed that she had an abnormally slender neck for so large a head. She takes after her father in general physique. He also has a large head with a small neck, wearing a 7⅜ hat, but a 14½ collar.

A number of other details may be added. The tongue is coated towards the root. Her breath in the night and before breakfast has been "bad" since her symptoms developed. Is largely a mouth-breather, like her father, though her mouth is closed part of the time in sleep. She is called full-blooded. She has a peculiar appetite, "literally living on bread-crusts, milk and cocoa," including pudding. Pupils equal, pulse 132 or more (standing). No helminthiasis. A slight cough the past week. Spleenic dulness not large. Always a great perspirer. Is thought to be a little feverish at times. Temperature 99.7 deg. Is slightly knock-kneed, but no costal knobbing.

The next morning after I first saw her, an interesting phenomenon made its appearance. The dorsal surfaces of the basal phalanges began to be puffy, tender, red and glistening. There was a distinct induration of each of these when seen three days later. Something of the same appeared about the lids of the left eye. This peculiar condition of local swelling, however, soon cleared up. It was too early to be any reaction to treatment, and was more likely some manifestation secondary to her general condition.

She was immediately put on thyroid, and has been kept on moderate doses ever since, except for about a month that fall, when a recurrence of symptoms made a return to thyroid necessary. At first she received three-quarters of a grain daily in divided doses. This was gradually run up to a grain and a half daily. Recently a reduction of this amount has been instituted. Improvement became evident in a few days, and inside of a week the previously necessary dose of bromide and chloral was discontinued for good. Her complexion soon cleared up. The peevish turns by day and the attacks by night entirely disappeared. Her mind cleared up and became active and despite thyroid medication she has kept fairly plump, in fact, has gained some six pounds in the past year. She now laughs most of the time, and is as well, bright, rosy and natural as any child. Mosquito bites in plenty last summer failed to start any dermatitis.

This spring she had an attack of tonsillitis, and stood it well. Her old physician and the parents are more than satisfied with the result, even though some thyroid medication has to be continued. Her own thyroid gland is still small, though a diminutive right lobe with scant isthmus and a stump on the left, all very soft, can now be made out. It seems to be regaining its function.

She still perspires too freely. There is an inclination to bronchitis, and the tonsils are large. The pulse continues over-rapid, usually 120, and faster if affected by any of the inevitable ailments of childhood. At times a small amount of strophanthus has helped, and some care as to laxatives is necessary. Of course she gets a liberal diet; though the appetite is fickle, she is "very fond of meat." It might be queried whether these present deviations from the normal are not due to the thyroid administered. According to Ausset, children are very susceptible to this agent. But the dose is small even for a child, and the deviations are only such as the patient had shown before taking the thyroid. Either animal thyroid does not fully substitute the native article, or certain minor symptoms in this child are not thus to be remedied.

Treatment was in this case based at the start on the view that the girl inherited through the father an abnormally small thyroid, and that this had become inadequate soon after she reached her third year. At that time she certainly appeared to be developing signs of myxedema, but that it was not the simple form was shown by the tendency to perspiration and the elevation rather than depression of body-temperature. It at least was not that alone.

These same facts, with the history of early bowel trouble, the later constipation, the age of the patient, her peculiar diet, decayed teeth, sedate manner, recurring touches of bronchitis, and the slight bending of the lower extremities (knock-knees, indicating late rickets) all point to a degree of rachitis. And the failure of the thyroid to remedy certain manifestations harmonizes with this view.

Many myxedematous patients, on the other hand, suffer from rachitis or some similar disorder (claimed by Siegert not to be true rickets). Various published cases show that we are here in a field where all kinds of mixed forms occur. And one can not but be struck, on looking up the literature, with the probability that many such cases as this have been called cretinism, myxedema, etc. We might call this case rachitis associated with a degree of athyreosis (to adopt a term from Quincke). The good result of treatment remains the same, under whatever head the trouble is classed.

Case III—Thomas P., 3 1-3 years old. Referred by Dr. Applegate, December 4, 1901. Father born in Brooklyn, mother in Minnesota, but of Irish race, boy in Montana. They moved here from the latter state in November, 1900. Since then the boy has been taking malt with hypophosphites, and improved for a time, but has recently retrograded. He has a brother of 5 years, and a sister older. The brother had convulsions when one year old, and again when three years old.

Tommy, the patient, was weaned when five months old. When but three days old he had a hemorrhage from the bowels, in which the blood streamed out for sixteen hours. During his life in the West, ever since he was small, up to the time of coming here, he had 16 to 18 watery, foul-smelling bowel-movements a day. Between the ages of one and three years he several times was threatened with convulsions, but they were warded off by the doctor. There was no thought of any serious trouble with him, however, up to last spring. At that time, in April, 1901, he had his first general convulsion. He has had four well developed ones since the last one five months ago. There were two partial ones only a week since, before getting up one morning. All of these came without warning. Besides these he has a lighter form, which may recur a dozen or twenty times a day. In them his head is drawn down on the breast, the eyes roll upward, and either his hands double under, or his arms go upwards. He will grasp at anything and try to work it off. Strikes his head "when it is over with" (head-knocking is recognized as a not unusual thing in rachitis). These lesser seizures have continued for some five months. Crying or excitement suffices to start one. They begin as he wakens about 6 A.M., occur again before rising, and there are often 5 or 6 in all before breakfast. He is a playful, uneasy little fellow; is said to be "awfully irritable and cross." Owing to large tonsils and a pug nose, he is a mouth-breather, though it is claimed that he sleeps with his mouth shut. Sleeps from 8 P.M. to 7 A. M., but is not sleepy by day. Maximum circumference of head 50 cm. Tongue clean. Is said to grow well. The fontanelle was very slow in solidifying, and did not fully close until after he was 2 years old. At the time seen there was a decided broad elevation at the site of the fontanelle. No paralysis anywhere. "Quite a cough, especially at night, quite a whoop," says the mother. With the cough recently there is also a bad odor to his breath. Weight scant 30 lbs., a gain of barely a pound in a year. Has a childish form of abbreviation of his words in speech. "He is crazy to eat all the time, and will eat anything."

Doubtless in this case all the attacks were of rachitic origin. He was placed on thyroid and syrup of the iodide, the previous treatment being continued as well. A month later, the father wrote me of "the complete recovery" of the boy. He says further: "The baby seemed to improve immediately, and in about a week the convulsive movements left him, and they show not the least sign of returning. He is also improved in every way." In April, Dr. Applegate reported that the result continued excellent, though the child was still taking thyroid. I saw him again this May. No return of the minor attacks. One "real

spasm" some time ago, from a gross error in diet. Has taken no medicine the last few weeks. He has now a rosy color, clear complexion, and bright, healthy look. Has gained 3 lbs. in weight (*i.e.*, in 5 months, to less than 1 lb. the previous year). The peculiar elevation at the fontanelle has disappeared, so that now there is only a slight fulness of that region. "He has grown much stronger on his feet." Mornings he still has a foul breath. The four upper middle teeth are little more than stumps. T. normal. P. 110, and apt to be fast.

While the symptoms in this boy, directly due to the rickets, yielded promptly and have not returned, it is still possible that the occasional full convulsion may prove to have become established. As the older brother had convulsions when about his age, but not since, and the patient has improved so well, it may fairly be hoped he also will now continue free.

A fourth case, seen in May, 1901, might be mentioned in which thyroid also acted admirably, but as it was very similar to the last, it is hardly necessary to give the details. In still another case of rachitic convulsions, seen before either of these, it is worth noting that the mother had a goiter (said to have followed measles).

While it has long been recognized that rickets may cause convulsions in the very young, the above cases indicate that obscure seizures, not strictly convulsive, may occur in children, from one form or other of disturbed general nutrition. And as evidence of such fact, this series of illustrative cases is, I believe, worth putting on record. It is chiefly to the rachitic element that the rest of my remarks will be directed.

As there appears to be much confusion regarding matters in this connection, it may not be amiss to point out some of the different questions here:

1. As to the irritability of the nervous system that may accompany rickets. This is now generally recognized. Nodding and rotatory spasms of the head, laryngospasm, etc., are largely of rachitic origin. Eclampsia infantum and the convulsions of dentition may often be due to the same cause. Tetany is another condition often associated with rickets; and what is more interesting here, it may also follow loss of the thyroid function. Thyroid feeding even proves successful in a proportion of cases of tetany—possibly those due to rickets.

2. Another, and less settled question is that of the causation

of epilepsy by rachitis. Gowers is about the only systematic writer who regards it as a prominent factor. Ohlmacher, formerly of the Ohio State Institution, has taken up this view, approaching the subject from the pathological side. He concludes that rickets plays an important part in the history of idiopathic epilepsy. My cases do not bear directly on this point.

3. It by no means follows that treatment directed to rachitis would be of value in any given case after true epilepsy had developed.

4. We now come to the use of thyroid in rickets. It has recently been noticed, though I have not the reference at hand, that some but not all the disturbances seen in rickets are relieved by thyroid. But I know of no cases like those described treated in this manner. They indicate that the severer forms of nervous disturbance, when directly due to rickets, are fully amenable to this treatment. So far the remedy has proven trustworthy, and in its action surprisingly prompt. The underlying trouble may not be cured, but its effects are so far ameliorated as to cause no further anxiety. In these cases its administration has not hindered the natural growth of the child, but favored it. The length of time that this remedy is to be kept up is a matter that must be determined in each case. To head off any tendency of rickets to induce epilepsy, the agent should be given in time and before a convulsive habit has been established.

Reports of the trial of thymus, suprarenal and ovarian extract in rachitis show them to have no definite value.

5. An allied question is that of the value of thyroid in epilepsy. The above cases do not bear on this point. The results obtained by Froehn and Hoppe (1899), were not such as to indicate any lasting benefit. And my own experience with it in several cases has been no more favorable, even though the patients had clearly suffered in earlier years from rickets. The use of thyroid in the eclampsia of pregnancy is, however, highly recommended by H. O. Nicholson[2], who makes a strong argument and supports it with the report of successful cases.

6. Are such cases as the above really epilepsy, and what would become of them if left alone? They seem to exhibit a

[2]Scot. Med. and Surg. Journal, 1901, June.

tendency to retrograde, at least as to frequency of attacks, though, perhaps, not as to severity. The longest case covers a free period, under treatment, of nearly two years. This may not include the possibility of something developing later, though it looks like a cure of the nervous disturbance. In any case the benefit received marks an advantage not to be secured in any other way.

In obscure cases of this kind, we are certainly warranted in making a careful search for signs of rickets or similar aberration in nutrition, and of making a trial of thyroid. For the present this remedy should be considered an essentially symptomatic one, and the other usual curative means should all be promptly instituted. To attempt any discussion of the various theories of the causation of rickets, of the claim that it is on the increase in America, of the relation of scorbutus to rachitis, of fetal rickets, etc., is beyond the purpose of this paper. Some of the points made, may, however, be summarized as follows:

1. In the young there occurs a class of cases characterized by recurrent attacks of heterogenous type, and that may conveniently be called "pseudo-epilepsy."

2. This form of trouble is curable.

3. Such cases, so far as here studied, are due to, or associated with, disturbances in the general tissue metabolism of the body.

4. Some of these are in whole or in part of rachitic origin.

5. Troubles of this kind when due to rachitis are amenable to thyroid treatment.

6. True epilepsy is not remedied by thyroid, even in a person who was once rachitic.

7. It is evident that in many cases there is a closer relationship between rachitis and athyreosis than has heretofore been recognized. There must be a relative inadequacy of the thyroid function in these cases associated with rickets. Either, as one of my cases indicates, there is a serious impairment of the activity of the gland, or thyroid feeding serves to burn up harmful material at large in the system.

A CASE OF TUMOR OF THE CEREBELLUM; AUTOPSY.

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On December, 19, 1900, Mrs. X., consulted me on account of a chronic cough and dizziness. She was sixty-four years old, had never been very strong and always more or less neurotic. This cough she had had for years, more or less exaggerated at times, with, lately, considerable expectoration. The condition of the lungs was that of a chronic bronchitis, and examinations of the sputum showed no tubercle bacilli. The cough and this bronchitis played no part in the future history of the case.

For four months she had had a great deal of palpitation of the heart and vertigo, these occuring synchronously. With this dizziness there was no ringing in the ears, no disturbance of the eyes, and no nausea. She also had no appetite and was very constipated. The temperature at this examination was 97.4°, and the pulse 96. She was unable to walk without swaying, and would fall if not assisted. This ataxic condition had been gradually developing during the last four months. There was and had been no headache, pupils reacted normally, patellar reflexes were normal. The urine was found normal with the exception of an increased amount of urates. The diagnosis was made at this time of some disturbance of the circulation in the cerebellum.

She was put to bed, and while strophanthus quieted the heart and the nutrition was maintained, no treatment of the cerebral condition was of any permanent avail. Her dizziness, which during the previous months was especially marked when she first lay down, also often occurred in the middle of the night without any cause or disturburance, or without even a dream; the simple fact of awakening would precipitate an attack. The result of bed-rest was a temporary improvement; the dizziness became less frequent, and consequently the attacks of palpitation were less frequent, as the latter occurred only at the time of the vertigo.

In February of 1901, that is in two months, the dizziness, which had become infrequent with the first rest treatment, had begun to again increase in frequency. She was utterly unable to sit up without becoming dizzy, and could not turn her head suddenly without an attack of vertigo. In the latter part of this month the dizzy turns became again less frequent, but more severe. She would lie all day with her head turned to the

right, claiming that she was dizzy if she turned her head to the left. This was without regard to light. The muscles of that side of the neck were slightly contracted, but the contraction could be easily overcome.

Soon after this the attacks of vertigo began to show epileptoid signs, and during the latter part of the month (February) there were typical epileptic convulsions. Just before this, during the attacks of dizziness, she had begun to complain of double vision, and objects would move and wave before her eyes. One month after she first went to bed she had begun to complain of ringing in the ears during these attacks of vertigo, beginning first, and at first only, in the right ear, but late in February there was ringing in both ears. During these attacks she now began to complain of intense pain in her head referred to the occipital region and described by her as of a "tearing apart sensation" with the sensation of "saw-teeth pricking."

When the epileptic attacks began the left arm was the first to move, and the body always turned toward the left side first. Her movements were frantic and repeated. There was more or less spasmodically voluntary clutching and tearing of the hair with both hands, and the expression of the face was that of intense pain and agony. There was no absolute loss of sensibility, as she would be apparently insensible and then immediately complain of the pain in her head. Inhalations of nitrite of amyl stopped these attacks. She would then sleep more or less soundly from a half hour to an hour. Large doses of bromides kept these attacks, at first, from occurring more frequently than every other day, but they soon became of daily occurence, and except for the inhibitory power of the amyl nitrite a status epilepticus would develop, she having repeated attacks during two or three hours with constant head-pain, and requiring several attendants to keep her from clutching her throat and tearing her hair. During these periods of her trouble the reflexes were all exaggerated, and the vision was perfect.

From this time on, *i.e.*, the latter part of February, she gradually failed, the epileptic attacks became more prolonged; they would come on very rapidly and the convulsive part was more prominent than in ordinary epileptic convulsions. She would thrash around the bed if not held, and as above stated would tear her hair, clutch her throat and throw herself out of bed if not held. She frequently gave sharp cries during these attacks. The eyes were sometimes staring, but from beginning to end there was never any paralytic eye-signs. She gradually ceased to complain of the ringing in the ears.

About March 4, 1901, she became rather stupified and slept a great deal. The bromide was discontinued, as there were

now no more epileptic attacks. She could be aroused to swallow or to speak, and recognized the people about her. At this time her eyes were carefully examined and no retinitis found, and the optic disks were normal in appearance and normally cupped.

On March 5 she could take no more food, and lay in pretty constant coma, in which she remained until March 8, when she died. During this semi-comatose period when she could be aroused she would sometimes cry because she said she was unable to say what she wanted to say, and would sometimes start to ask me something and once said that she knew what she wanted to ask but was unable to tell me what it was. Previous to this period she had complained of incoördination in that she said she would attempt to turn to one side of the bed and find herself turning to the other side. She would start to take something with her right hand and find herself taking it with her left. Up to the very last day that she lived her pulse was always good, and she died without having had any Cheyne-Stokes respiration.

There was no paralysis of the sphincters. During her last days there was a good deal of superficial venous congestion, so much so that her face was very dark, as were also her extremities.

My diagnosis had been made at the time the epileptic convulsions began of tumor of the cerebellum, more on the right side than on the left, which diagnosis was confirmed by Dr. Francis Bacon.

We were not allowed a general autopsy in this case, but were permitted to open the skull and examine the brain. The autopsy was performed by Professor Charles J. Bartlett. We found the brain normal except that the right lobe of the cerebellum was distinctly larger and firmer than the left on account of a tumor within it. An examination under the microscope showed this tumor to be a glioma. This growth was sufficient in size to give the pressure symptoms which the case presented. I inferred that the tumor was on the right side, because the ringing of the ears began on that side, and the cortical irritation from the pressure caused the convulsions to begin in the left arm. I expected to find a considerable amount of fluid in the third ventricle. We did find some fluid, but perhaps not more than the normal amount.

CHICAGO NEUROLOGICAL SOCIETY.

December 19, 1901.

The President, Dr. Hugh T. Patrick, in the chair.

The Relations of the Areas of the Various Levels of the Spinal Cord in Cross Section.—As a result of studies made in the laboratory of the University of Chicago by Mr. Davis concerning the relations of the areas of the various levels of the spinal cord in cross section, Dr. Donaldson made the following report. He said that a number of spinal cords had been examined and an average length obtained. With this average length as a base line curved lines were drawn in a parallel direction representing first the variation from it produced by tracings of the spinal enlargements, and second variations from it produced by tracings from the axial lines of the gray and white matter. In other words, curvilinear lines were drawn in the same direction as the base line, showing the relations of various levels of the cord *in toto* and also the relations of sections of the gray and white matter in progress through the cord.

The old plat which appeared in Quain's Anatomy and other textbooks was copied from the original work done by Stilling. His chart was made from sections of a five-year-old child's cord. It has appeared in the literature since 1874 without mention of the fact that the chart was made as indicated. As yet no comparison can be made between the child and the adult cord as the work is too new.

The claim is made for this sort of work that by a comparison of the area of the cross section of any given spinal cord with the corresponding area of the standardized cord knowledge might be had as to the condition of the cord compared with the normal.

Dr. Hugh T. Patrick inquired whether a piece of foil cut out to correspond to the gray matter was used as the basis of measurement.

Dr. H. H. Donaldson said that the section was first enlarged about four times, the outline then drawn on tracing paper which is placed over the foil, which is cut in accordance with the figure. From that figure the calculation is made. The foil used for this purpose must be of uniform thickness so that the measurements will be accurate. The foil is sold in strips and as the ends of the strip are much thinner than the middle piece, only the latter is used. These strips are all weighed so that they will correspond to the standard. The measurements can also be made with cardboard.

Dr. Hugh T. Patrick thought that this subect was of some practical importance in some of the degenerate diseases, especially cerebellar ataxia, in which the central nervous system has been found very small. He could not recollect any statement bearing on the small amount of white or gray matter, but simply recalled mention of the small size of the brain, the cerebellum, pons, medulla oblongata and cord. In one case measurements of the cord were given in different diameters. He was of the opinion that in connection with some of the congenital diseases these comparative measurements might be of considerable interest.

Dr. H. H. Donaldson saw a possible use for these measurements in the conditions mentioned by Dr. Patrick. It would enable one to state

how much a given section deviated from the standard. He said that although the total area of white and gray matter might be a little larger or smaller, the relative development was very well maintained.

Dr. L. F. Barker inquired as to the kinds of reproductions in Stilling's Atlas.

Dr. H. H. Donaldson said they were lithographs and were probably pretty accurate. The cross sections of the cord were placed in cells filled with alcohol. The observations were made while the specimens were in a good state of preservation, before the alcohol had shrunken them.

On Growth Changes in the Ganglion Cells and Dorsal Root Fibers from Different Segments of the Spinal Cord.—This paper was read by Dr. H. H. Donaldson. Dr. Donaldson said that the results presented were based on work which had been done in the laboratory by Mr. Hatai, who is a fellow in the department, and who, at Dr. Donaldson's suggestion, undertook a study of the growth changes in a certain class of nerve cells. This class being the cells constituting the dorsal ganglia of the spinal cord.

The animal used was the white rat. Ganglia were taken from three different segments of the cord of each animal used. The ganglia examined were the sixth cervical, the fourth thoracic, and the second lumbar. The animals selected for study were four in number. The weight of the animals was ten grams, twenty-four grams, sixty-eight grams, and one hundred and sixty-seven grams, respectively. The ten gram rat was about five to eight days of age. The largest, about six or seven months old. At about the sixth month rats begin to reproduce, so that the largest one had already reached the age of reproduction. We have then spanned between the smallest of these rats and the largest concerned a period extending from shortly after birth to maturity. In each of these animals three spinal ganglia and the connected dorsal nerves were examined.

The first point investigated was this: Is the number of the nerve cells constituting a spinal ganglion constant from birth to maturity? The figures which were obtained for the cervical ganglion were as follows: In the rat weighing ten grams there was found something over ten thousand of these cells. In the rat weighing twenty-four grams, over nine thousand cells. In the sixty-eight gram rat, over twelve thousand, and in the hundred and sixty-seven gram rat over eleven thousand.

The differences in these numbers were interpreted as individual variations occurring in the number of cells in spinal ganglia in which there is no decrease or increase,—that is in the sense of production of new cells,—during the growing period. Dr. Donaldson thought it might be fairly inferred from these observations that the number of spinal ganglion cells remains constant during this period. That is an important point to settle. This statement is not, however, equivalent to a statement that no changes are taking place in these ganglion cells during this period. As a matter of fact all the cells are growing, that is, increasing in size.

Furthermore, the recent studies of Hatai on the spinal ganglion cells have shown that there are two classes of cells. They may be divided into the small and the large cells. Under these circumstances it was of interest to determine whether the relative number of small cells was the same at maturity as at birth. So as not to overburden his hearers with this mass of figures, Dr. Donaldson used simply those for the sixth cervical region in a diagram which showed the ratio of large to small cells. There was in this ganglion a certain number of small cells and large cells which footed up to the total. Dr. Donaldson called attention to the fact that these numbers are variable, the large cells steadily in-

creasing in number, whereas the small cells drop off in number as the rat increases in weight. Thus, if we make a ratio of the small cells to the large cells, we find that in the youngest rat there are something over 3.4 small cells for every large one. At the next stage three small cells for every large one, at the third, two and three-tenths cells for each large cell, and in the last rat one and four-tenths cells for every large one. In other words, with the increase in age of the animal the number of small cells steadily diminishes and the number of large cells is correspondingly increased.

In the ganglia of the thoracic and lumbar regions the same changes occur. The next point touches the number of nerve fibers in the dorsal nerve roots. Our usual representation of the spinal ganglia is to draw a single ganglion cell on the board and ask our audience to multiply this unit and make a ganglion of it. This typical ganglion cell always has two branches, one of which passes to the spinal cord, as the dorsal nerve root fiber, and the other towards the periphery, in the peripheral nerve. If this completely represented the structure of the ganglion then we should have as many fibers in the dorsal nerve root as we have cell bodies in the spinal ganglion, because each ganglion cell giving rise to one branch forming the dorsal nerve root fiber would be represented in the root by one fiber, and in the ganglion by one cell body. From studies, both on mammals and on frogs, we know that every time the attempt has been made to determine the relation between the number of cells in the ganglion and the number of fibers in the root, there has been found a large excess of ganglion cells in the ganglia. Observations were then directed to finding out the exact relationship between the two.

The total number of the dorsal root fibers was shown in a diagram for the cervical, thoracic and lumbar nerves. Dr. Donaldson said that in the cervical root the number when smallest was 1,998, increasing to 4,227 when largest, and the same thing appears in each one of the other columns. Thus if you take a rat of ten grams, he will have in this locality 1,998 fibers for that particular dorsal nerve root, whereas a mature rat has 4,227 fibers in the same nerve root. In other words, the number of fibers in the dorsal root has materially increased, between the ten gram stage and 167 gram stage. This is a new point as regards the mammal. As a frog grows larger the fibers in the dorsal nerve root increase in number. Thus while it has been known for a long time that this takes place in the frog, it is new to the mammal, but if it holds good in the frog and in the rat it will most probably hold good in man. In the rat at birth, then, we have one fiber where at maturity there is something over two. In the thoracic region we find one fiber at birth to two and a half at maturity, and in the lumbar nerve one fiber at birth to two and one-tenth at maturity. It holds good then in all of the different levels that the number of fibers is at first small and steadily increases as the rat grows larger. The suggestion at once arises that we may associate this increase in the number of fibers with the increase in the number of large nerve-cells in this ganglion. In the cervical region the number of fibers is nearly equal to the number of large cells. It is 1 fiber to 1.2 large cells; 1.092, 1.097, and 1.1 for the four rats chosen.

In the thoracic and lumbar regions there are nearly twice as many of the large cells as there are fibers, so that according to this observation the cervical differs from the thoracic and the lumbar ganglia in this relation of the number of the large cells to the number of fibers. The ganglia are differently constituted.

Now to return to a consideration of the number of cells in the young ganglia as compared with the number of fibers in the corresponding dorsal

root, and the relation of these two during the growth process. For every fiber in the cervical dorsal root there are at birth 5.5 cells, which number gradually diminishes until it drops down to 2.7 cells at maturity. Not half the cells are in commission, in the sense of furnishing dorsal root fibers, in the cervical ganglion of the mature rat. The discrepancy is much more marked in the thoracic and lumbar regions. They start with 11 cells to every fiber and end up with about 5 cells; ending where the cervical began. In every instance there is a tendency for the cervical ganglion to be precocious and more complete in its development.

We have then verified completely the fact of the excessive number of cells in the spinal ganglia. We have shown that that excessive number decreases gradually as the number of nerve fibers in the dorsal root increases with the growth of the young, and that at maturity there are in the rat anywhere from 2.7 to 5.7 cells in a spinal ganglion for every fiber that exists in the dorsal nerve root.

These are the principal points which Dr. Donaldson wanted to make evident although others were shown on the diagram, but they did not bear on these two fundamental points. First, the constancy of the number of nerve cells in the spinal ganglion throughout life. Second, the progressive increase of the number of nerve fibers in a dorsal nerve root. Third, the disparity between the number of fibers and the number of cells, that for the fibers standing to that for the cells as 1 to 2.7 or 5.7 in the several cases.

Dr. Donaldson spoke as to the possible application of these results. They were also studying the activity and growth of intelligence in the rats. They found that rats which are of about this weight, 24 grams, and which have therefore about one-half the total number of nerve fibers they are destined to have in a dorsal nerve root, are the most active. When they get larger they become slow and comparatively inactive. In other words, as far as this particular animal is concerned, the very active period is correlated with the period in which the afferent roots are only half completed. The animal has still to double the number of nerve fibers in the dorsal root, and while that is going on it becomes a much less active animal than it was in the early stage.

Dr. Donaldson believed there is every probability that this condition of things will be found to obtain in man. They had no spinal cords at present to work on and hence are blocked in their studies. Although it is impossible at the moment to correlate these periods of development with the period of development in the growing child, he would say that between 25 and 68, somewhere about 30 or 40 grams would correspond to a six or seven-year-old child. If the relations in the development of the nervous system are the same, we would then find the child with incomplete dorsal nerve roots at a time when it is about as active as it ever will be. The settlement of this question must await the appearance of suitable material.

Dr. L. F. Barker regretted that Dr. Donaldson did not divulge his opinions based on the results of this work. Naturally a great many questions arise. He was especially interested in getting an explanation of the reason for the increase in the number of fibers. Three possible explanations suggest themselves. One, the growing-out of more fibers from the cells in the ganglion to the cord, probably the most likely explanation. The second possibility would be a division of single fibers already present, and the third an outgrowth of fibers from the cord to the ganglion, although that might be the least plausible.

The second point was the disappearance of so many of the small cells, the increase in the large cells, which apparently was due to the development of the small cells. Nevertheless there are in all cases a large

number of small cells left undeveloped. He said he would like to know whether these were surplus cells that never attained to maturity, remaining small cells without any processes, or whether they possibly can be cells which send out axis cylinders to end in cell bodies of other neurones. If they remain immature it would be in accord with what is believed to occur elsewhere in the nervous system, namely, that in all parts of the nervous system, as in most parts of the body, there are cells left in an immature state capable of further development under certain circumstances. If that is the case there are as many reserve cells in the ganglia as there are cells in commission. That would be a very large excess of reserve cells, but if it is remembered that as in the substantia gelatinosa many cells develop and degenerate, as many as attain maturity, the excess would not seem so great. He also asked for the reason of the increase in the ratio of fibers to cells, especially in the cervical region. Naturally the first thing to which attention would be directed is the possibility of the enormous development of the hand as a sense organ as compared with the thoracic region, and the lower extremity as a sense organ. He did not know whether in the rat the hand was more of a sense organ than the foot. In the human being we might naturally expect a greater number of root fibers in the cervical roots than in the thoracic or lumbar. The hand area in the cerebral cortex is very large compared with the trunk or foot area. Another point, that this change in proportion takes place during the point of greatest activity of the animal, is extremely interesting, and suggests the possibility of a relationship between activity and development. It is a well-known fact that the exposure to strong light of prematurely-born cats increases the rapidity of development of the myelin sheath. Why could it not be possible that the further activity should determine this rapid change in the ratio of fibers to cells? He hoped that Dr. Donaldson would explain these points.

Dr. Donaldson said that we might account for the increase in the number of fibers in the dorsal nerve root by assuming that the new dorsal root fibers grow out from cells which entered into commission in the later stage of the ganglion, being immature in the early stage. That explanation receives its best support from the relation in the cervical region between the large cells and the number of fibers. In the dorsal nerve root the number of large cells is equal to the number of nerve fibers. The division of fibers does not appear so probable in the sense in which it would have to take place, although many of the fibers are divided. These divided fibers all pass to the periphery, but such a division has always been observed at a stated place, the dividing branches being of equal length and equal growth, and do not simply represent a series of twigs which might run out and thus add to the number of fibers. All the evidence tends to point toward the throwing into commission of new cells for the addition of new fibers to the dorsal nerve root.

The next question, as to the excessive number of cells in the spinal ganglia, he deemed a rather hard nut to crack. There were a few cells, the axones of which ended within the limits of the spinal ganglion itself. These, however, are few in number and cannot account for the very large apparent excess of the cells in the ganglia. The excess will probably be found to be due to the immature cells which have not yet sent their axones out any great distance. The higher one goes in the nervous system, the greater appears to be the excess of cells found in the different localities. In man the excess is probably much greater than in the frog or rat. That is a rather dangerous thing to say because it is in the nature of a prophecy. However it does not appear to be impractica-

ble. It is rather difficult to say what is the function of these cells and yet there appear to be many of them in the cortex and there is no reason why they should not appear in the spinal ganglia. It seems to be a property of the nervous system to contain more parts than it actually needs and lives up to.

As to the difference between the number of fibers and the number of cells in the thoracic and lumbar regions as compared with the cervical region, he believed that possibly the sympathetic nerves have something to do with this. He said his first impulse was to associate the excessive number of cells in the thoracic region with the sympathetic connection which is absent in the cervical. This, however, is but a poor explanation, as it is open to question. As to use and disuse, something might be made out of that suggestion, even though there is nothing at present that would bear directly on this. He suggested that rats might be brought up in loneliness and solitude where activity would be reduced to a minimum, and then be compared with rats brought up in playful activity, so as to determine whether there is any difference in the development of the spinal ganglion cells.

Dr. Hugh T. Patrick inquired whether the fibers in the brain had ever been counted. Whether there is a great increase in the fibers with the mental and intellectual development of the individual, or whether they simply take on sheaths. Whether the corpus callosum at birth contains more fibers than in the adult.

Dr. Donaldson said that he had found the fibers present, but they did not have any medullary sheaths. The cell-bodies might be present, he said, but not their axones. He felt that the axones are a later growth in a large number of the cell elements. These observations have been made only from the standpoint of medullation, showing the increase in medullation as maturity is reached. He was positive that the axones are not present in rats. The rat's cerebrum can be cut very deeply and a few weeks later it is impossible to find the scar. The wound grows over completely, because the brain is cut at a time when most cell elements are in a neuroblast stage. There are no axones. For that reason it is difficult to find the place of injury.

Dr. J. W. Engbert said that from comparative anatomy and biology it has been ascertained that the cord at one time had different segments which seemed to be more or less similar. In man it is found that certain segments gain a great deal, as, for instance, the cervical and lumbar, whereas the thoracic loses. In what does the gain consist? Do the segments gain chiefly in cell bodies, or do they send out fibers?

Dr. Donaldson said that in the gray substance, where it is voluminous, there is an excessive number of cells. There is also an excessive number of fibers as is shown by the rise in the curve in the white substance coincident with the enlargement of the gray. The white substance is distributed both around the gray matter and within it. In the lumbar region the gray matter is comparatively less than the cervical enlargement, and in that sense the smallness of this segment is really a reduction in the amount of white matter in it. It is accomplished by change in the shape of the cells. They lie like watermelons, on end, in the thoracic region; on their side in the cervical; and in the lumbar region as if they were set on their side with the widest plane in the plane of this section. They have different shapes according to the segments in which they are located. Though the segments are short in the lumbar region the amount of gray matter is as large as in the cervical segment.

Dr. L. F. Barker asked if the base lines were made so that the seg-

ment distances were equal, how, then, would the gray matter lie.

Dr. Donaldson said that the vertical length would have to be multiplied by the horizontal length, and the difference in the area exaggerated by making the segments equal. There is a relative increase of gray matter in the two enlargements when reduced to cubic contents.

March 20, 1902.

The President, Dr. Daniel R. Brower, in the chair.

A Case of Hysteria.—Dr. Elbert Wing reported the case of Miss H., aged fifteen years, who complained of a constant ache in all of the teeth of the upper jaw, accompanied by tenderness in all of them; both the aching and tenderness being greatest on the right side. The pain is described as a dull, heavy ache, rather than sharp, cutting or lancinating, varies somewhat in severity, and is present all of her waking hours. The tenderness is not great and not uniform in all of the teeth. The pain is influenced somewhat by cold, but not at all by heat. It is not increased by the acts of talking or eating. In May, 1901, the patient had an attack similar to this, in which the teeth of the lower jaw were involved. It began in a few of the teeth on the left side. Dr. F., her dentist, drilled into these, destroyed the pulps, and filled the root canals. This did not stop the pain, and finally the affected teeth were extracted. This treatment with variations ran the circuit of her lower jaw, until all of its teeth were extracted, and a plate of artificial teeth put in. About six weeks after the teeth were all extracted, the pain, diminishing gradually, ceased. Six weeks after all pain ceased in the lower jaw, it began in the teeth of the right side of the upper jaw, and had continued until this examination was made, December 21, 1901. The dentist did not excavate or extract the upper teeth.

Three years ago the patient had what she described as "soreness in her scalp," which was not relieved until her hair was cut short. Then she had some trouble with her eyes, not inflammatory, in which she says she "almost lost her sight." Later she had attacks of spasms. She says that one doctor stuck pins in her and said that "there was nothing the matter with her." She has had no other illness, and menstruation is fully established, regular and normal in every way.

The patient's father has had "nervous trouble," is now well, and the family history is otherwise negative.

Examination shows a young woman, five feet eight inches tall, large in proportion, in excellent general nutrition, and full mammary development. Appetite good; no symptoms of indigestion; bowels act normally, and sleep is good, except when disturbed by the pain in the teeth. Loss of sleep from this cause, she says, is considerable. Patient's appearance is that of excellent health. When alone with the examiner the patient's manner is quiet and free from peculiarity, except lounging in her chair, but when her dentist, who seems familiarly acquainted, is present, her manner is petulant and capricious. The thoracic and abdominal organs are normal.

Voluntary motor power is normal and symmetrical. Tests for touch and pain were made with camel's hair brush, and common pin. Sensation to touch is slightly less throughout the left side. On this side there are areas of moderate size of diminished sensation over the upper half of the chest, the hypochondriac region, the outer and middle third of the thigh, and two places on the leg. There is absence of pain to the prick of a pin over and just above the left

breast, and there is diminished pain sense in the areas partially anesthetic to touch, and slight anesthesia over the left scapula. Epigastric reflex is slightly less on left side; abdominal walls symmetrical; elbow-jerks are absent; knee-jerks are present and fairly symmetrical. The pupils react normally, and vesical and rectal control are reported normal. There is moderate tenderness in the upper teeth, more pronounced on the right side, and some in the upper branch of the fifth nerve of the right side. There is no other tenderness. Physical examination is not carried further. The patient was referred to her home physician with the diagnosis of hysteria and the usual outlined suggestions for treatment.

Dr. Harold N. Moyer asked whether the patient had a mania for operations, to which Dr. Wing replied: "It would seem so."

Dr. Charles L. Lodor inquired whether there were any anesthesia of the mouth.

Dr. Wing replied that there was not.

Dr. Lodor asked whether the teeth were sensitive or not.

Dr. Wing replied that they were not.

Dr. Sydney Kuh recalled the case of a girl who presented herself at Czerny's clinic in Heidelberg, with the statement that she had swallowed a pin. She had considerable pain; her stomach was opened, and a pin was found in the wall of the stomach. The patient returned subsequently two or three times, wanting to undergo another operation. The suspicion of the surgeon was aroused; he refused to operate a second time as he was firmly convinced there were no more pins in the stomach.

Dr. Kuh also detailed a case of hysterical deception which occurred in the practice of a country doctor. The country practitioner sent the specimen to Dr. Edmund Andrews for examination, with a history that the girl had swallowed a snake, that she could feel it moving about in the abdomen, and insisted that something be done for its removal. But little or no attention was paid to her until one day they found her in front of the house quite ill apparently. She had vomited and in the vomit there was a snake-like body, which was sent to Dr. Andrews for analysis, and it turned out to be the gut of some animal.

Dr. Daniel R. Brower mentioned a case which occurred in the practice of Dr. Fitch many years ago. It was stated that the woman's urinary secretion had been entirely suppressed, and that she was secreting urine by the gastric mucous membrane. He was called in consultation in this case. Dr. Fitch had the contents of the woman's stomach examined a number of times, and always found urine in the contents. He suggested to Dr. Fitch that she be put to bed and some responsible person be directed to watch her. This was done, and it was found that the woman would urinate, and then swallow the urine.

Dr. Lodor referred to a patient who had some of her teeth extracted on account of a supposedly distressing condition of them. She exhibited the usual stigmata of hysteria. She was referred to him with the recommendation that the dental nerve be trephined for excessive pain; but on examination he found nothing wrong with the patient's mouth, and said the case was purely hysterical.

Periscope.

Rivista di Patologia Nervosa e Mentale.
(Vol. vii, fasc. 6, June, 1902.)

1. Anomaly of the Cerebellum. D. Della Rovere and B. De Vecchi.
2. Complete Ablation of the Thyro-Parathyroid System in Dogs Treated with Halogenated Fats. G. Coronedi and G. Marchetti.
3. Contribution to the Pathogenesis of Exophthalmic Goiter. E. Tedeschi.

1. *Anomaly of the Cerebellum.*—A minute description of what is believed to be the first case on record of division of the worm into two distinct lobes.

2. *Complete Ablation of the Thyro-Parathyroid System.*—Six dogs, subjected to thyro-parathyroidectomy were treated with bromated fat (dibromostearic acid). In most of the cases it was administered before the operation; in one it was continued after the operation, and in another administration was solely post-operative when the symptoms induced were at their height. The effect of such treatment was attenuation and final disappearance of the phenomena caused by the operation, or their entire absence for a more or less prolonged time. Not only was this true of nervous and muscular excitation and depression but also of the dystrophies of cachexia, the result being prolongation of life in a condition of more or less perfect health. Three dogs subjected to the same operation were treated with chloro-iodostearic acid and two with diiodostearic. In the former, results were most satisfactory; in the latter, hypodermic administration, as used in one instance, alone gave good results. The authors believe that the salutary influence of the halogens studied, upon animals subjected to complete ablation of the thyro-parathyroid system may be attributable not alone to the symptomatic effect of Br. and I., but to a more complex physiological action, representing within certain limits the functions of the thyro-parathyroid tissue; and that, should this theory be proven, the experiments cited constitute a valuable contribution to the biological functions of Br. and I. in the physiology of the thyro-parathyroid system.

3. *Contribution to the Pathogenesis of Exophthalmic Goiter.*—A previous series of experiments having shown that lesions of the restiform bodies provoked typical symptoms of Basedow's disease, the author has sought to ascertain the relation of the thyroid to such manifestations by increasing or diminishing the thyroid function in animals in whom lesions of the restiform bodies had been induced. The work is thus summarized: (1) in animals, lesions of the restiform bodies (especially of their anterior portion) produce marked symptoms of Basedow's disease; (2) in animals in whom the symptoms of Basedow's disease have been induced by lesions of the restiform bodies and in whom such symptoms have greatly diminished or disappeared, they may be reproduced wholly or in part by producing in them a condition of hyperthyroidization; (3) in animals in whom the thyroid has been removed, lesion of the restiform bodies does not cause symptoms of Basedow's disease; (4) in animals in whom the symptoms of Basedow's disease have been induced through lesion of the restiform bodies, removal of the thyroid diminishes or causes the disappearance of the greater part of such symptoms. R. L. Fielding (New York).

Revue Neurologique.

(Vol. 10, 1902, No. 9, May 15.)

1. Benedikt's Syndrome, Single Tubercle of the Cerebral Peduncle. L. Astros and E. Hawthorn.
2. Tic and Function. Henry Meige.
3. Contribution to the Psychophysiology of the Dying. Two Cases of Chronic Chorea. N. Vaschide and Ch. Vurpas.
4. Generalized Atrophy of the Musculature of All the Viscera in a Progressive Amyotrophy of the Aran-Duchenne Type. André Léri.

1. *Benedikt's Syndrome.*—The authors consider this affection to be not as rare as has been thought and cite many cases. They give the history of what they consider an absolutely typical case, concluding, as follows: The clinical symptoms observed in life were so characteristic that diagnosis was easily given of tubercle of the right cerebral peduncle. The incomplete character and limitation of the lesion were shown at autopsy, as the lesion extended from the pons to the optic thalamus. Paralysis of the right oculomotor began with ptosis, then by progressive extension became total, although incomplete for certain muscles; the pupil was not in complete mydriasis, and for some time responded to light stimuli. The incomplete oculomotor paralysis has been noted in many cases and indicates that the lesion has not affected the nerve at its extrapeduncular emergence. The pupil was occasionally myotic. The integrity of the fibers from the superior nucleus indicated that the lesion did not extend to the foot of the peduncle, and that it must be localized in the upper part, where the fibers of the oculomotor nerve are disassociated. It seems there must be peduncular hemorrhages of inferior as well as superior position. In one, hemiplegia is complete, followed by permanent contracture, without disturbance of sensation or trembling; here oculomotor paralysis is usually total. The lesion is situated in the lower part of the peduncle. In the second type paralytic symptoms are at a minimum; this contracture, if existing, is variable; sensory trouble sometimes noted; characteristic trembling is present; oculomotor paralysis partial and incomplete. The lesion is superior and at first there is only compression and excitation on the pyramidal fibers. This second type can not remain pure on account of the extension of its lesions.

For a pathogenic interpretation of the trembling, there must be excitation of the fibers of the pyramidal fascicle by a contiguous lesion, not of a serious lesion destructive to this fascicle. Lesions of the superior position must be more apt to bring this about. There is the question, however, whether a subjacent inferior lesion, a meningitis of the base, could by irritation of the pyramidal fascicles, cause the same symptoms.

2. *Tic and Function.*—Tics, such as they are, may be considered as *perturbations of functional acts.* The following considerations are given in this connection: First, it is necessary to define *function.* There are certain functions, as circulatory, digestive, excretory, taking place without control of the will, and certain motor phenomena are dependent upon special muscular systems. Disturbances of these functional phenomena do not come under tics. Other functional movements executed by striated muscle are controlled by will. Among these motor functional acts, certain, as those of respiration, are vital, and as such must not be confused with the regulative functions. Others, as nictitation, masti-

cation, locomotion, are indispensable and common to all. Others, as salivation, are most useful to individuals, but not universal. Other acts, as writing or swimming, are acquired and termed functional, though they are not indispensable to life nor common to all. Thus the term is very comprehensive and covers many grades; so we look to the principal characteristics of functional acts. In all there is repetition, periodicity. External causes or will may modify these acts, as breathing, and there are individual differences; but there is nevertheless a norm, conforming to the natural law of the least effort, and to this there are no exceptions but pathologic cases, and it is these that are studied. If a subject disobeys the law of the least effort for the functional end, by excess or by incompleteness, there is functional perturbation. A large number of tics are simply this, functional acts which exceed or fall short of the end normally to be attained. Another mode of perturbation is execution of a functional act at the wrong time, as a smile, cry, word, when there is no natural suggestion for them; for function has another characteristic, that it must be preceded by a need and followed by satisfaction of it. Some professional acts are termed functional, and when disturbed, as in cramps, spasms, and professional impotence, may come under the heading of tics. But though most tics are made evident as well by nothing as by anything definite, professional cramp takes place only during the performance of the professional act.

There is still another class. Those functions made out of whole cloth under dominance of an unreasonable idea for an absurd end. Many tics are of this order; true they may be characterized by repetition, need and satisfaction, but the repetition is too frequent and too violent, the need imperious, the satisfaction disproportionate, all abnormal, and this function is inopportune if not dangerous. It is evident, then, that the appearance of a tic denotes imperfection in the mental state, particularly the will. Clinical observation confirms this point of view.

3. *Chronic Chorea at Death.*—The authors, after detailed report of the cases, conclude with the following considerations on their observations during the period of agony: (1) In chronic chorea characterized by continual and purposeless movement, which even sleep does not arrest, the movements cease completely some days before death, in the present cases four days before. The patients resumed their habitual expressions and attitudes; a period of calm appeared to precede the final cataclysm; (2) the patient, although with well-defined anatomical lesions, according to many authors, can not, theoretically at least, act otherwise before death by reason of some indefinable mechanism; the pathological physiognomy is cut off and the patients acquire the function of their automatic movements perfectly, reflexes and volitions. This fact would militate against lesions, or else there would be a curious resumption of function; (3) the observations point in favor of the concomitance of the cessation of these continuous movements and the disorganization of the phenomena of consciousness. In other words, there is an intimate relation between the comatose state which precedes death and the changes due to the psychophysiological troubles of the patients. He falls into a state of coma, ceases at the same time to have the tics, keeping a calm physiognomy and a normal attitude, with an unmistakable mental change. Thus in one case characterized by excessive pathological emotion, weeping and groaning, the patient on cessation of the movements two to four days before death was absolutely passive.

Thus the observations explain, if only to a degree, the psychophysiology of the mental state, going partly to make up the phenomena known as *presentiment of death.* There is a paradoxical state in the troubles due to anatomical lesions which ceases suddenly by reason of approach of death. Conclusion beyond the facts themselves is impossible, but solution of them in the future is to be hoped.

4. *Atrophy of the Visceral Muscles.*—After detailed reports illustrated by cuts of the viscera affected, the conclusion is reached that today there can be held paramount one single fact: a myelopathic muscular atrophy need not be confined to the striated muscles of voluntary life, but may affect also all the visceral musculature, the smooth muscles of organic life; in this case the stomach, intestine, bladder, gall bladder, ductus choledichus and heart were all atrophied, not altogether, but merely in the muscular layer.

It is not known whether the lesion reported is or is not exceptional. In the case the atrophy dated sixteen years back, and perhaps a longer one is necessary to produce amyotrophy of the viscera capable of troubling noticeably their functions and of drawing attention to them at autopsy. To this view the authors are inclined after noticing extreme atrophy of the heart which not until the last months produced only a little arhythmia. The asthmatic respiratory crises, repeated during the last months of the disease, suggest the question whether visceral amyotrophy (heart, muscles of Reissessen) does not play an important rôle in the pathogeny of at least certain "bulbar crises" which so often end the lives of amyotrophics. Other functional troubles also due to visceral amyotrophy will doubtless appear in the future, and already P. Marie has noted in certain amyotrophics persistent constipation, which perhaps would not be independent of intestinal amyotrophy.

JELLIFFE.

The Journal of Mental Science.

(1902. April, Vol. 47, No. 201.)

1. The Treatment of Incipient Mental Disorder and Its Teaching in the Wards of General Hospitals. SIR JOHN SIBBALD.
2. Hallucinations and Allied Mental Phenomena. SIR LAUDER BRUNTON.
3. Hospital Ideas in the Care of the Insane; a Statement of Certain Methods in Use at the Stirling District Asylum, Larbert. GEORGE M. ROBERTSON.
4. Some Further Remarks upon Night Nursing and Supervision in Asylums. FRANK ASHLEY ELKINS.
5. The Bearing of Recent Research in the Posterior Root Ganglia upon the New Theories Concerning the Etiology of Tabes Dorsalis. R. G. ROWS.
6. A Flaw in the English Lunacy Law. ERNEST W. WHITE.
7. Mental Conditions Resulting in Homicide. G. T. REVINGTON.
8. Clinical Notes and Cases. Abnomal Brain of Excessive Weight, J. SUTCLIFFE; With Pathological Report, S. DELÉPINE; Tumor of Frontal Lobes of Cerebrum, T. P. COWEN.

1. *Incipient Mental Disorder and Clinical Teaching.*—The great desideratum of more thorough clinical teaching of psychiatry prompts this able paper of Dr. Sibbald. It is a question peculiarly appropriate for American psychiatry as well. The author points out that Griesinger was the leader in the attempt to provide more adequate clinical instruction in mental disorders. As far back as 1866 he induced the authorities to permit such teaching, which has continued to the present day much amplified. This teaching is now a part of almost every medical curriculum, but is for the most part carried on in asylums, at great distance at times from the medical schools, and with consequent bother and annoyance to both teacher and student. A more vital question, however, is that of the importance of hospital treatment for the early stages of mental disorder in the interest of the patient and quite inde-

pendent of question of clinical instruction. Such wards were first introduced in the Glasgow poorhouse hospital by Dr. Carswell some twelve years past and have done signal service.

Dr. Sibbald gives some suggestions as to the manner in which such wards might be constructed. He speaks of the reasons why patients with incipient mental disease have not received proper hospital treatment and why thus many presumably curable insanities have become hopelessly chronic. The reasons are due to statutory restrictions and to social considerations this latter being one of the banes of present knowledge, or rather lack of it, of diseases of mind on the part of the laity. Thus legal and social reasons are responsible for this neglect.

The author proposes the setting apart of certain wards in the general infirmary for the treatment of these incipient cases, and lays emphasis on the value of the general treatment as advocated by Weir Mitchell in the treatment of these affections. He also refers to the more universal employment of women nurses in male wards, such as is seen in Copenhagen, for instance.

In respect to the clinical teaching the important fact is emphasized that such teaching given in the asylum is of little service to the practitioner since asylum cases are not the kind he is going to see; it is the incipient cases he must learn to recognize if he is to be of service, and these he never sees in such an institution; therefore the greater need for the wider use of treatment of the incipient insane in general hospitals.

[The paper is of importance and worthy of being read in full, especially in view of the New York movement for the establishment of Psychopathic Hospitals as planned by Dr. Peterson, President of the Lunacy Commission. J.]

2. *Hallucinations and Allied Mental Phenomena.*—Dr. Brunton here presents a very interesting paper dealing with a large number of different ideas. Many of these are popular among those interested in psychical phenomena and while little new is added much that is old is conveniently collected under a comprehensive heading. He shows that which is now very widely held by practically all students of psychological phenomena, that many of the heretofore considered abstruse and incomprehensible manifestations of nerve activities are dependent largely upon the greater amplitude of perfection in the special senses of some individuals. The workers in psychic research showed these things to be true some years ago. Dr. Brunton still would believe in divining rods, because, apparently, the explanation of their activity does not come within his ken. This subject has been most ably and exhaustively handled by others. The theory of hallucinations set forth on retinal or other changes affecting a sense organ are interesting but not unique. The article, while not bearing abstraction because of its diffuseness, makes extremely interesting reading.

3. *Stirling District Asylum Methods of Hospital Management.*—This paper must be read in its entirety to be appreciated. The ideals here set forth tempt one to the expression of the feeling that should they become mentally affected the Stirling Asylum would be a most desirable place for treatment. The gradual awakening of the spirit of doing something for the insane is exemplified in the rationale of hospital treatment here outlined. One of the most important features in the article is the thorough advocacy of female nursing for both men and women. This subject is here very ably dwelt upon.

4. *Night Nursing in Asylums.*—Dr. Elkins considers questions of nursing administration holding in the main that: (1) the night arrangements in asylums should closely approximate those existing in general hospitals; and (2) all acute noisy, dirty and destructive patients

be placed at night in dormitories under constant supervision, and be removed only when it is evident they have ceased to require special care. As to the number of patients that can be looked after at night by one nurse, it is suggested that in a ward for acute, feeble and sick patients, the limit should be placed at twenty-five, while in a dormitory for quiet patients, requiring raising on account of their habits or attention during a fit, the number might be about forty or fifty. This paper also pleads for more female nursing for males.

5. *Tabes and the Posterior Root Ganglia.*—Dr. Rows, pathologist to the County Asylum, Whittingham, summarized the theories concerning the pathology of tabes particularly in relation to changes in the posterior root ganglia. He summarizes the theories showing that the hypothesis had lost favor within recent years. Scriuti's modification of the original idea is discussed at length. This Italian observer has said that the degeneration of tabes consists in changes in fibers which have started with some defect and which degenerate under the influence of some stimulus; and the lesions which are found on examination will depend on the situation of the fibers which have been injured by this congenital or acquired lesion, which fibers will, in later life, degenerate if they be attacked by some poison such as syphilis, alcohol, pellagra, etc. This hypothesis is rivaled by that of many modern workers, who maintain that vascular changes are all important in the etiology of tabes and other degenerative conditions. The author does not give any new light on these hypotheses, he simply states them.

6. *Flaw in the Lunacy Law.*—A case of suicide due to the ignorance of the legal authorities as to what constitutes insanity.

7. *Mental Conditions Resulting in Homicide.*—Dr. G. T. Revington gives a summary of the histories of some forty-nine male patients who have been admitted to the Dundrum Asylum charged with murder or with manslaughter. Wives, he says, are the commonest victims, then children, then parents and children. Mothers-in-law seem to be immune. Homicide is stated to be a potentiality in all cases of delusion and hallucination, and that whether a homicide occurs or not is not so much a matter of peculiar mental condition as a matter of environment, a matter of length of time that elapses before safety is sought within the walls of the asylum. Eight of his patients had acute frenzy, twelve were under the influence of alcohol, ten had acute hallucinations and in ten there was mental blankness. He sums up saying that he believes that his homicidal cases did not suffer from any form of mental disease. Most of them he did not consider criminals in any sense of the word, crime being an accident in a mental disorder, not an essential or typical outcome. None of his cases led him to believe in a homicidal mania, a ravenous lust for blood, a brutalized craving to take life simply for the sake of taking life. Ordinary motives such as jealousy, misery, acute fear acting on morbidly active emotional conditions are responsible, he believes, for the lengthly list of murders.

8. *Clinical Notes and Cases.*—(*a*) Report of case of brain weighing 69 ounces, with microscopical examination showing it to be hypertrophy of brain by reason of diffuse increase in neuroglia and with gliomatous masses—a myxomatous glioma. (*b*) Tumor of frontal lobe with sleep persisting eight months. The sleep appeared to be natural, the appearance of patient like one overcome by fatigue. He was easy to awaken, would answer rationally for a short time, and then with waning interest would fall asleep, even while eating. Optic neuritis was the only other objective symptom. Sarcoma of frontal lobes was found post-mortem. JELLIFFE.

Monatsschrift für Psychiatrie und Neurologie.
(1902. April, Vol. 11, No. 4.)

1. Laugh-Stroke (Laugh-Vertigo, Laugh-Swoons). H. OPPENHEIM.
2. The Relation of Hysteria to Epilepsy. O. KAISER.
3. A Rare Form of Crises in Tabes Dorsalis and Tabetic Crises in General. OTFRID FOERSTER.
4. Pathological Anatomy of Paralytic Dementia. THEODORE KÄS.
5. Experiment of Psycho-physiological Representation of Sense Perception. STORCH.

1. *Laugh-Stroke* (Laugh-Vertigo, Laugh Swoons).—Oppenheim reports two similar cases of paroxysmal laughter or "Lachschlag." In both at the height of laughter caused by some joke or witicism the patients suddenly lost consciousness for a few seconds and fell to the ground without convulsions. The author concludes that the pathology of such attacks is unknown and that they deserve a separate classification from epilepsy.

2. *The Relation of Hysteria to Epilepsy.* Kaiser presents a critical essay in defense of the recent position taken by Steffens that transitional cases between epilepsy and hysteria still deserve the old title hystero-epilepsy in the older meaning of the term as first used.

3. *A Rare Form of Crises in Tabes Dorsalis and Tabetic Crises in General.*—Foerster gives a careful analysis of the different forms of the sensory and motor types of tabetic crises with their pathology, and presents notes of cases of "extremity crises" consisting of paroxysmal clonus or tonic movements. Subjective sensory symptoms may or may not precede or accompany such phenomena of motor discharges. The clonic or tonic convulsive movements constituting the form of crises is most frequently seen in the legs. They have an analogous pathogenesis to those of the sensory type, namely, a neuronic irritation and destruction of the reflex arc and its collateral paths in the cord.

4. *Pathological Anatomy of Paralytic Dementia.*—Continued.

5. *Experiment of Psycho-physiological Representation of Sense Perception.*—Continued.

L. P. CLARK.

Neurologisches Centralblatt.
(1902. May 16, No. 10.)

1. Demonstration of Cortical Sight. E. HITZIG.
2. On Acute Cerebellar Ataxia. W. v. BECHTEREW.
3. Failure of Knee-reflex in Dorsal Compression Myelitis with Degeneration of the Posterior Roots in Lumbar Cord. M. BARTELS.
4. Further Over Asthenic Paralysis. S. GOLDFLAM.

1. *Cortical Visual Area.*—From experimental operation on the cortical visual area in dogs, Hitzig advanced the following: (a) that after slight unilateral operations the visual disturbances disappear in a shorter or longer time; (b) this disappearance, like the visual disturbances, at first occurs in the median and under half of the field. After bilateral operation on the cortical visual apparatus he obtained the following results: (a) with two exceptions, the second operation produced a return of the visual disturbances of the first; (b) this disturbance was as intense as in that of the first instance; (c) this disturbance increased. In some cases, it was worse some time after the operation than on the second day; (d) circumscribed section as Munk describes was not observed; (e) the visual disturbance, after some time disappeared.

2. *Cerebellar Ataxia.*—A further communication on a syndrome

noted in the *Neurologisches Centralblatt* in 1900, by the same writer, consisting of a cerebellar gait, vertigo, Romberg, following coma, or ordinary sleep. This was considered to occur in chronic alcoholics, but the case reported by Schweitzer, due to gastro-intestinal intoxication, leads von Bechterew to call attention to the fact that he suggested that etiological factors other than alcohol might cause the symptoms.

3. *Knee-reflexes and Spinal Compression.*—A study of a case of Pott's disease with complete paralysis and loss of knee-jerks. A practically complete transverse lesion was forced at the fourth dorsal segment. The posterior roots from the second lumbar segment to the middle of the sacral cord were found degenerated. He considered the lesions of the posterior roots to be the result of mechanical causes, *i.e.*, congestion of the cerebrospinal fluid, and suggests lumbar puncture in incomplete pressure myelitis with loss of reflexes.

4. To be continued.

McCarthy (Philadelphia).....

MISCELLANY.

Therapy at Farnham House. By W. R. Dawson (Dublin Journal of Medical Science, July, 1902).

During the past year seven patients were subjected to thyroid treatment. One case only, a recurrent melancholia, showed a direct benefit from small dosage. Several times when an attack had begun, five grains three times a day seemed to mitigate the symptoms and shorten the attack. A case of subacute mania got 375 grains in 6 days and made a rapid recovery, and a similar case of six months' standing received 530 grains in 9 days with prompt recovery. Both cases became very fat after the treatment. A case of climacteric melancholia with delusions was given 345 grains in 7 days without effect. Some improvement followed 160 grains in four days in a paranoiac. Slight glycosuria in one case, slight albuminuria in another, and a vomiting attack in another were the only untoward effects of the treatment. Thyroid would seem to be indicated in states with high arterial pressure, especially melancholia and stupor. On the contrary suprarenal is most useful in conditions of excitement and exaltation with low blood pressure, as in acute mania of fairly recent origin without stupor. Suprarenal was not good in a case of acute confusion with high blood pressure, no good results were obtained from the administration of 30 grains of dried gland a day for 3 days, 45 grains for 7 days, and 60 grains for over 3 weeks in a rheumatic subject with acute mania and high arterial pressure.

Erythrol tetranitrate was tried in severe high pressure cases, but in only one, a senile mania with delusions and stupor, did it seem to act well. Nitroglycerin gr. 1-50 three times a day diminished epileptic seizures after bromipin had lost its controlling influence. Bromipin acted favorably on the whole, but sometimes seemed to be depressing. Substitution of paraldehyd for bromipin was followed in 10 days by an attack of ephemeral mania such as had not occurred for three years. The attack subsided quickly under potassium bromide and ergot. Paraldehyde was of doubtful benefit in melancholia, but with potassium bromide is a good soporific and seems to increase the appetite. A convenient vehicle for this drug is glycerin and water strongly flavored with cinnamon.

W. A. Bastedo.

Book Reviews

Les localisations fonctionnelles de la capsule interne par Jean Abadie." Bordeaux. Imprimerie G. Gounouilhon, 1901.

The study of the finer anatomy and physiology of various portions of the internal capsule is far from being complete. The conclusions at which various writers arrived in their investigations in regard to the function of some parts of this important structure of the brain are so contradictory that any new contribution on the subject will always be of interest.

Dr. Jean Abadie's book is interesting and instructive from several standpoints. To make the subject clear he divided his work with good reason into five chapters. First of all he gives a complete historical review of the researches made in the domain of the internal capsule. We learn that Burdach (1819-26) was the first who created the name "internal capsule." The true scientific study of the subject begins with Türck (1853), but Charcot in 1872 was the first to give a detailed anatomical and physiological description of this region. It was Charcot who traced the boundary line between the pyramidal and sensory bundles in the internal capsule. A long series of writers follows. One after another investigated the function of various portions of the internal capsule and all arrived at the following subdivision: the anterior portion which is psychic, the middle which is motor, and the posterior which is sensory. Soon each of these localizations was subdivided and multiplied. Some of the last divisions proved to be incorrect and some doubtful.

In the anatomical portion of his work Abadie presents a criticism of the most important sections of the brain which give the best results for studying the internal capsule in all its aspects. They are those of Vicq d'Azyr, Pitres, Brissaud, Flechsig and Dejerine. The relative value of these sections are explicitly criticized: the advantageous and disadvantageous points are mentioned and discussed. Finally the author describes the classical anatomical division of various tracts of fibers which the internal capsule is composed of.

In the chapter on physiology the author refers to the various experiments relating to the function of the internal capsule, experiments which led to negative results until Fritsch and Hitzig in 1810 discovered the four motor centers of the cortex by means of the galvanic current. Ferrier in 1873 corroborated the existence of the cortical centers and first investigated the function of the capsular region; but under the last name he understood the white fibers, the thalamus and corpora striata. His experiments therefore did not have a direct bearing upon the capsule itself. It is only from 1877 that real work upon the physiology of the internal capsule was conducted; in France by François-Frank and Pitres, in England by Beevor and Horsley for motor function, and by Laborde, Lemoine, Sollier and Verger for sensory function. The motor portion is represented in two anterior thirds of the posterior segment of the capsule. The anterior half of it as well as the basal ganglia are not excitable. As to the sensory portion of the capsule it is only since the work of Sollier and Verger in 1898 that real conclusive experiments were conducted. Others before them speak of sensory disturbances only in regard to cortical lesions. The last two observers prove conclusively that a lesion of the excitable portion of

the internal capsule (median and posterior segments) causes an incomplete motor as well as a sensory paralysis of the opposite side of the body.

In the fourth chapter Abadie considers separately the various tracts of the internal capsule: he gives a critical review of experiments and observations made upon the pyramidal tract, the fibers coming from the knee of the capsule, the anterior portion of the capsule and finally upon the sensory portion, the existence of which is now so much disputed. As to the bundle causing hemichorea the author shows that any portion of the pyramidal tract can produce this symptom. The capsular bundle for aphasia he does not accept, but believes in dysarthria which, however, may be due to a lesion in the anterior portion of the capsule. In Chapter V the author relates 22 cases, the pathological findings of which he discusses in detail. The most interesting statement made by him is that he could not find a single case of disturbance of general sensation and special senses with a lesion strictly limited to the internal capsule.

In regard to association of motor and sensory paralysis he reports cases in which other portions were affected except the posterior third, also cases with total hemianesthesia of the Türck-Charcot type, while the lesion was at the apex of the lenticular nucleus.

In conclusion the author expresses a doubt in regard to the function of some portions of the internal capsule, but speaks with emphasis of the constant association of motor and sensory paralysis. According to him there is no distinct bundle in the internal capsule, a lesion of which would cause hemianesthesia without an accompanying motor paralysis.

The book is very useful for its impartial and clear exposé of different views held by prominent neurologists upon one of the most important subjects of cerebral physiology. ALFRED GORDON.

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Original Articles.

PARADOXICAL PSEUDO-HYPERTROPHY FOLLOWING INFANTILE CEREBRAL HEMIPLEGIA.

By L. Pierce Clark, M.D.
NEW YORK.

The hypertrophy of paralyzed parts in infantile cerebral palsy is a rare condition. No special article on the subject exists in literature, and no cases illustrative have been reported in America, although several observers, such as Sachs, Peterson and Osler have made diligent search for them. The different degrees of muscular and bone atrophy in spinal palsy of children have been well studied and satisfactorily explained on a basis of a disturbance in the trophic center of the anterior horns of the cord, the site of the destructive palsy lesion. Notwithstanding the almost uniform presence of atrophy in spinal palsies (acute anterior poliomyelitis) Sellegmuller has described a bony elongation in spinal palsies, and Dejerine claims to have demonstrated in such cases true fiber hypertrophy in those muscles totally paralyzed. Joffroy and Achard have also proven the truth of Dejerine's position in spinal palsy cases, even 40 or 50 years after the original onset of the lesion. However well the positive exclusion of a few nerve cells from the destructive lesion in the cord might satisfy the hypothesis that by their overaction fiber hypertrophy might occur, no such explanation could be urged for the hypertrophic condition found in cerebral palsy cases, as there are no trophic centers in the brain as such. If such

functions are exercised by the brain they must be through muscular movements represented in the cortex. A normal musculature in cerebral palsy cases is common and easily explained on the theory of a slight or transitory brain involvement and intact anterior horn cells of the cord, but the case assumes perplexing aspects in the extreme when actual hypertrophy of paralyzed parts occurs after the initial paralytic stroke.

Brief Historical Review—The voluminous literature of infantile cerebral palsy deals largely with the condition while the patients are still in infancy, and as hypertrophy probably requires some years for its development we cannot expect to note many recorded cases. Moreover, but 30% to 50% of essential palsies develop epilepsy, therefore less than one-half of adult cases come under careful observation, and then too for epileptiform attacks usually. The existence of the latter symptoms often fails to excite the clinician's attention to the initial organic mischief, as hopes of ameliorating a lesion so long existent are not often entertained. Then, too, most cases of hypertrophy are found in literature on athetosis, notwithstanding in the twelve recorded cases (five my own) but eight had athetosis.

In the pioneer study of hypertrophy in athetosis, Hammond in 1871 describes no case dating from infantile cerebral palsy or from childhood even. Clay and Shaw also failed to find hypertrophy in his careful study of choretic paresis in 1873, but Bernhardt has the honor of describing the first case in 1876, the same year in which Gowers also reports a case, or possibly two, although the occurrence of the second is doubtful. In 1878 Oulmont, in a thesis on athetosis, includes one case of hypertrophy, and Goudard described one case in 1884 in a general article upon cerebral palsies in children. In an extensive article on athetosis, Berger in 1885 stated that a small percentage of adult athetosis show the state of hypertrophy, which he believed to be an inexplicable freak of pathology. In 1891 Schieber reported an interesting and minutely-described case, and Kaiser, in 1897, recorded a unique case of marked hypertrophy of muscles and bone, complicated by myotonia. The

chronological order of the cases of hypertrophy on record, including three reported for the first time in this paper, are as follows: 1876, Bernhardt, 2, athetosis present; 1876, Gowers, 1, athetosis present; 1878, Oulmont, 1, athetosis present; 1884, Goudard, 1, athetosis present; 1891, Schieber, 1, athetosis present; 1897, Kaiser, 1, athetosis present; 1900, Clark, 2, athetosis and epilepsy present in one; epilepsy without athetosis present in the other; 1902, Clark, 3, epilepsy present in all without athetosis.

The family history of the hypertrophic form of infantile cerebral palsy presents no marked neuropathy, although in cases reported by Bernhardt, Schieber, Kaiser and myself, convulsive disorders such as epilepsy, tic disease and chorea were found in the collateral family stock.

The intra-uterine life of the patient has proven uneventful in all cases except some slight and insignificant emotional disturbance in the mother. Labor has always been at term and no evidence of trauma or severe asphyxiation has been found to exist. Instrumental delivery was practiced in two cases, but no injury resulted. All the children were heathful until the time of the stroke, except in two who developed infantile eclampsia at dentition. The cases of hypertrophy are equally divided between the two sexes. The age of onset of the paralytic stroke in cases in which hypertrophy developed later ranges as follows: two at 18 months; three at four years; three at six years; three at seven years; one at fourteen years; an average of six years of age, considerably later than in ordinary palsy cases (under 3 1-2). The causes ascribed for the palsy in the 12 cases have been: four, scarlet fever, or its sequela; two, errors of dentition; and in six none, as in ordinary palsy cases. The immediate excitant to the stroke in all cases was too trivial for consideration.

Usually the more important symptoms of the onset of cerebral palsy have been absent in the hypertrophic cases, but in all of mine there were more or less severe convulsions, vomiting, fever and physical prostration. In two cases reported by Bernhardt and Kaiser the palsy occurred without apparent knowledge of it on the part of the patient. The

presence of palsy was manifest on awakening in the morning. Usually there was considerable prostration following

Case I. E. P., paradoxical pseudo-hypertrophy in infantile cerebral palsy. Relative enlargement of right shoulder, arm and thigh. Right arm (paralyzed) at biceps 3-4 inches larger than left or well side. Left handed. Epilepsy (partial) and athetosis.

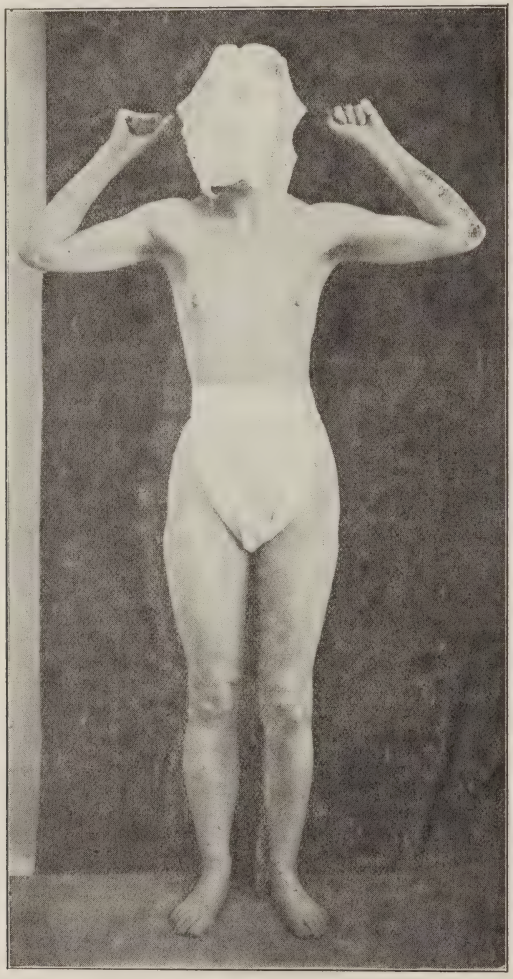
the stroke and the patients were bed-ridden for several days, weeks or months. The preferment of the hemiplegia for the right or left side is relatively the same as that for ordinary

palsy (7 to 5) in favor of the right side. The extent of paralysis has always been hemiplegic. The leg and face recover

Case II. F. W., paradoxical pseudo-hypertrophy in infantile cerebral palsy. Relative enlargement of entire right side, including face. Hypertrophy most marked in right arm 3-4 inch. Deltoid and biceps most hypertrophic. Epilepsy (partial) but no athetosis. Left handed.

most rapidly and completely, as is usual in ordinary palsy cases (a few days to several weeks). Athetosis was present in eight cases; its onset occurred on or before the patient re-

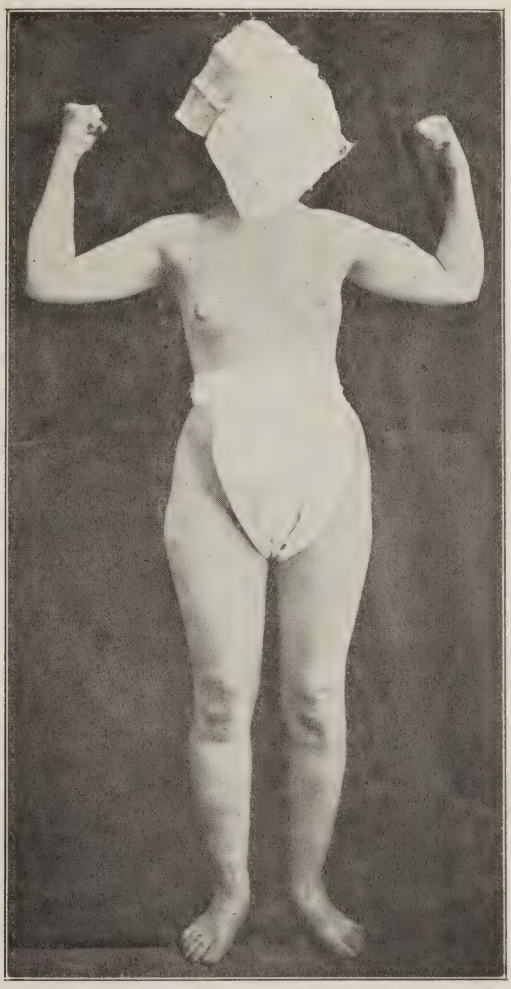
covered from the prostration of the stroke (choreic paresis) but in the two cases reported by Schieber and Gowers, athe-



Case III. J. P., paradoxical pseudo-hypertrophy in infantile cerebral palsy. Relative enlargement of left shoulder and arm (paralyzed); arm and left shoulder 1-2 in. larger. Muscles hypertrophic; deltoid, biceps and triceps. Forearm and lower extremity of the same side markedly atrophic (1-2 to 1 in.). Epilepsy (partial) but no athetosis; right handed.

tosis occurred in the one five weeks after the cerebral lesion, while in the other it developed one year after the stroke (post-

hemiplegic athetosis). The character of the spontaneous movements are generally described as typical of Hammond's



Case V. A. V., paradoxical pseudo hypertrophy in infantile cerebral palsy. Relative enlargement of left arm (hemiplegic side) 1-4 in.; hypertrophy confined mostly to biceps. Atrophy of forearm and lower extremity of left (paralyzed) side (1-2 to 1 1-2 in.). Epilepsy (partial) but no athetosis; hight handed.

athetosis (post-hemiplegic athetosis or chorea), but frequently it resembles choreic paresis (fibrillary chorea); the associ-

ation of the two forms are often seen in the same case. The movements are most marked in the forearm and shoulder muscles. In about one-half the cases the lower extremity engages in the athetosis in a minor degree, although in Bernhardt's second case the leg alone was the seat of athetosis and hypertrophy. The athetosis ceases usually in a position of rest or while asleep. Voluntary effort at control may decrease the movements, rarely it increases; voluntary acts bring on increased athetotic movements. The posture of the arm is usually that of partial rest with hand pressed against the side and elbow slightly extended, although the posture and movements, as in essential athetosis, may vary greatly.

The mental state of the patients on the whole is better than usually obtains in ordinary palsy cases. While the intellectual element as such is quite lacking, the patients exhibit considerable ability in learning industrial pursuits. Those cases in which athetosis exists and those in which it does not occur are about equal in ability to undertake trades. Possibly the non-athetotic are slightly duller students, but this defect offsets the physical agitation of the affected parts in the athetotic class.

The length of time necessary to produce the hypertrophy is obviously difficult to ascertain, but the time elapsing between the palsy lesion and the detection of the hypertrophy has been very variable, from 8 to 29 years, the average being 16 years. The age of the patients at the time the hypertrophy has been recognized ranges from 12 to 36 years, the average being 22 years. The hypertrophy may involve any and all parts that are paralyzed, even the breast and testicles may enlarge. The skin, muscles and fat may all hypertrophy in a single case, although some degree of muscle hypertrophy is uniformly present in every case. The enlargement has had its seat in the upper extremity in every instance except in Bernhardt's second case, where the leg was hypertrophic. The arm has shown most enlargement in nine cases, the forearm in two and the calf in one. The muscles most frequently hypertrophied are found in the following order of occurrence: biceps, deltoid and triceps.

The parts not hypertrophic follow the general law for atro-

phies in ordinary cerebral palsy cases. The hypertrophied tissues have not been studied carefully from the histological standpoint in any cases except in two of our own; in these true fiber hypertrophy existed. An enlargement and increase in the number of the muscle spindles was found; usually hypertrophy and hyperplasia went hand in hand. No doubt many of the reported cases of enlargement of an extremity should be classed as fat hypertrophy, as noted in a case by Gibatteau, a fact also known to Cazauvielh. In such cases the excessive fat appears to be compensatory for the muscular atrophy, as has been shown in poliomyelitis (Freud). Our suspicions are further strengthened inasmuch as the hypertrophic extremity is without exception relatively weaker than its opposite on the healthy side (pseudo-muscular hypertrophy). Too much stress, however, should not be laid on this fact, as the contractile power of any muscle in different individuals is not always in definite ratio to its volume. The difficulties of examining excised muscular tissue for fiber hypertrophy, either from the living or dead subject, have been well emphasized by Batten, Spencer and Gowers. In most cases the skin and fatty tissue have shown hyperplasia of their essential elements[1].

Bony hypertrophy was first actually proven by me in one of my cases (1899) by X-ray examination, and still more recently Lannois and Fayolle have reported the same results by the same method. The bones of the entire upper extremity may be hypertrophic, although the bones usually found enlarged are those corresponding to the hypertrophic soft parts which they underlie. The degree of circumferential enlargement of the hypertrophic extremity varies from 1-4 to 7-8 in., although not usually more than 1-2 inch. As to the cause of the condition. In the first eight cases athetosis was found, being most marked in parts hypertrophic, it was therefore logically inferred that the condition was due to the violence of the spontaneous movements.

I have, however, reported four cases of paradoxical hypertrophy in which no athetosis has ever existed, but in which

[1]A detailed histo-pathological report of the excised hypertrophic tissues is in preparation.

a severe partial epilepsy was present instead.[2] If the palsy lesion is slight or transient, the nutrition and integrity of the cortical elements remain more or less intact[3], and a post-hemiplegic disorder of motility, such as athetosis or a focal epilepsy, produces in time a hypertrophic condition. The essential factor is the extent and character of the cortical injury, otherwise we would find hypertrophy with every case of choreic paresis or partial epilepsy following palsy lesions. Indeed, extreme atrophy most frequently occurs after choreic paresis or focal epilepsy of hemiplegics. Therefore the association of the peculiar cortical lesion with some degree of post-hemiplegic disorder of motility in parts once paralyzed causes in time the paradoxical pseudo-hypertrophy.

REPORT OF THREE CASES OF PARADOXICAL PSEUDO-HYPERTROPHY IN INFANTILE CEREBRAL HEMIPLEGIA.

Case III. J. P., female; aged twenty-four years, maternal grandfather had chronic rheumatism and was alcoholic. Paternal aunt had idiopathic epilepsy which developed at the menopause. Patient is second in line of birth. The mother had one miscarriage and died of paralysis at fifty-eight years. Pregnancy and labor were normal. There was no paralysis or injury of child at the time of birth. As soon as the patient was able to get about she developed a depraved taste for plaster, clay, muddy water, etc. At 18 months old, during difficult dentition she had a series of convulsions which lasted 12 hours, immediately after which complete left hemiplegia was noted. Nine months from the onset of the hemiplegia the fingers of the left hand developed "cramping spasms" without loss of consciousness, at the time patient commenced to use the left side learning over again how to creep and walk. One year after the hemiplegia she had "falling attacks," attended by a sudden twitching in all the muscles of the left side, during which consciousness was frequently preserved. Patient is feeble-minded; has never attended school but can read and write. The epilepsy probably dates from the hemiplegia, although typical grand mal did not develop until she was 13 years old, during a period of severe menstruation. A sensory aura of numbness on the left side precedes attack. The seizures consist of tonic and clonic convulsions which begin and end on the left (paralyzed) side,

[2]The epilepsy may also be superimposed upon an athetosis, as in case E. P. of our series.

[3]It is well known also that partial epilepsy and athetosis most frequently occur after slight and transient lesions of the cortex.

as in ordinary spasmodic hemiplegia. Aside from the order of beginning and ending and the abbreviation of the post-paroxysmal stertor, the seizures are typical grand mal in character. The patient also has frequent petit mal, which usually occurs in the morning before rising. The attacks consist of "writhing movements" of the extremities, in which the body and limbs are "thrown in every direction." Grand mal attacks are infrequent (2 or 3 a month), while petit mal attacks occur every three or four days. She has had 25 grand mal attacks in 24 hours. Knee-jerks are exaggerated on the left side and there is considerable loss of power in the left arm and leg. The tongue is deviated to the right on protrusion. Weight 100 lbs., height 5 ft. 2 3-8 in., circumference of left arm (paralyzed) is 1-2 in. larger at biceps than that of the right, while the left forearm is 1-4 in. larger than the right. There is no athetosis or disorder of motility other than the epilepsy in hypertrophic parts and the left face and leg are atrophic. The condition of the hypertrophy is well shown in Fig. III. The measurements are as follows:

	Right	Left
Dynamometric test	44	22
Length from acromion process to external condyle	11 in.	11 in.
Length of internal condyle of humerus to the styloid process of ulna	9 1-2 in.	9 1-2 in.
External condyle to styloid process of radius	9 1-4 in.	9 1-4 in.
Circumference of arm, arm flexed on forearm at right angles	9 in.	9 1-2 in.*
Largest part of forearm in its anatomical position	7 3-4 in.	8in.
Circumference of hand at metacarpophalangeal articulation	7 in.	7 in.
Length of index finger	3 7-16 in.	3 7-16 in.
Length of second finger	4 in.	4 in.
Length of thumb	2 1-2 in.	2 1-2 in.
Circumference of middle finger at middle part of first phalanx	2 in.	2 in.
Circumference of thigh at gluteal fold	20 3-4 in.	20 in.*
Circumference at popliteal	12 in.	11 1-2 in.
Circumference of leg at calf	12 1-2 in.	11 1-4 in.
Dorso-plantar circumference	8 in.	8 in.
Circumference of wrist	5 1-2 in.	5 1-2 in.
Circumference of ankle	7 1-2 in.	7 1-2 in.
Length from ant. sup. spinous process to int. malleolus	31 in.	30 in.

Case IV. A. B., male, aged twenty-three years. Father is intemperate, mother had periodic facial neuralgia and epilepsy; she died from puerperal eclampsia at my patient's birth. Pregnancy and labor were normal in all respects. Patient is the youngest in a family of seven; all the patient's brothers and sisters are well. He always had migraine until his epilepsy began at 14; at this latter age complete left hemiplegia occurred, which was supposed to have been caused by overeating. He had convulsions during the stroke and was prostrated for several months from the physical exhaustion. The left side participates most in the classic grand mal epilepsy which occurs frequently in such cases. The vaso-motor disorders which occur ordinarily in palsy cases are found here also. Although the patient is able to do considerable work with the left hand, there is, nevertheless, a temporarily increased weakness in the left side after convulsions (exhaustion paralysis). Some permanent contractures exist in the flexors of the fingers. No athetotic movements have ever occurred. The power in the paralyzed parts is greater than on the sound side; all reflexes on the left side are exaggerated; dynamometric test shows: left, 48; right, 44. Weight is 136 pounds; height, 5 ft. 3 in. The left arm at the biceps is hypertrophied, the enlargement is 1-4 in. more on the paralyzed side. The remainder of the left side is atrophic.[3]

Case V. A. V., female, aged 36. One maternal aunt was tubercular; another maternal aunt died of epilepsy, and a maternal cousin also died insane. Both parents are very neurotic; the mother still has periodic attacks of neuralgia. The father had epilepsy from "18 months until five years old." A sister of our patient had convulsions at dentition at two, and after a respite of ten years epileptic convulsions began at twelve. She finally died of epilepsy at twenty-seven years of age.

Our patient was born a healthful child at full term. She was, nevertheless, a very "restless and irritable" child. She had scrofula, pertussis and measles. At seven years of age she had scarlet fever, which was followed by nephritis. A few days after the renal complication she had repeated convulsions, high fever and vomiting, which symptoms were followed by a comatose period for an hour. On return to consciousness the entire left side was found paralyzed. Epileptiform attacks began at once. At first the attacks existed as slight "fainting spells" in which the patient fell to the left if standing or sitting.

[3]Owing to the patient having passed from our observation after the first examination, we have not been able to furnish a more complete history or a pictorial reproduction of the case.

After a period of five years of hemispastic seizures, classic grand mal developed with the order of invasion and ending in the muscles of the paralyzed side. A sensation of heat rising from the feet to the head constitutes the aura. She has had ten seizures in twenty-four hours; she now averages one attack a month. No athetosis has ever existed in this case. The muscles of the left arm are hypertrophic, the biceps is most hypertrophic, as is well shown on page 647. As the biceps forms almost the entire hypertrophy of the left arm, the relative enlargement in this case, although but 1-4 in., appears much greater than in J. P., in which it was 1-2 in. All the tendon reflexes are much exaggerated on the left side. Weight, 120 1-2 pounds; height 4 ft. 1 3-4 in.; asymmetry of face, palate and cranium, which are development errors due to the hemiplegia rather than stigmata of degeneration, as ordinarily understood. The measurements of the case are as follows:

	Right	Left
Dynamometric test	52	19.5
Length from acromion process to external condyle	10 3-4 in.	10 3-4 in.
Length internal condyle of humerus to the styloid process of ulna	9 1-2 in.	9 1-2 in.
External condyle to styloid process of radius	9 1-2 in.	9 1-2 in.
Circum. of arm, arm flexed on forearm at right angle	10 3-4 in.	11 in.*
Largest part of forearm in its anatomical position	8 3-4 in.	8 3-4 in.
Circum. of hand at metacarpo-phalangeal articulations	7 1-4 in.	6 1-2 in.
Length of index finger	3 9-16 in.	3 5-16 in.
Length of second finger	4 in.	3 7-8 in.
Length of thumb	2 1-2 in.	2 5-16 in.
Circum. of middle finger at middle part of first phalanx	2 1-4 in.	2 3-16 in.
Thigh at gluteal fold	22 in.	21 in.
Circum. prosstitial space	14 1-4 in.	14 3-16 in.
Circum. of leg at calf	13 1-2 in.	12 1-4 in.
Dorso-plantar circum.	8 1-2 in.	8 7-16 in
Circum. of wrist	6 in.	5 7-8 in.
Circum. of ankle	7 7-8 in.	7 3-4 in.
Length of ant. sup. spinous process to int. malleolus	31 in.	31 in.

The following is an abstract of the two cases I have previously reported[4].

Case I. E. P., female, age twenty-three years; neurotic history. Although she was born a healthy child at term, labor was difficult and was finished by instruments. She walked at two and had no serious illness until six, when she had severe and prolonged convulsions for 24 to 36 hours. This left the right side completely hemiplegic. Epileptiform crises developed at once and have occurred every two or three days since. They are grand mal, involving the right side first and most. She is feeble-minded. Marked athetosis exists, being most marked in the right arm. The presence of athetosis dates from the day of the stroke. The deep reflexes on the right side are all exaggerated. The following measurements and Fig. I. show well the hypertrophy in this case:

	Right	Left
Weight, 138 lbs., Height, 5 ft. 3 3-4 in.		
Dynamometric test	33	60
Length from acromion process to external condyle	12 in.	12 1-2 in.
Length internal condyle of humerus to the styloid process of ulna	9 1-2 in.	9 1-2 in.
External condyle to styloid process of radius	10 in.	9 1-2 in.
Circumference of arm; arm flexed on forearm at right angles	10 1-4 in	9 1-2 in.
Largest part of forearm in its anatomical position	9 1-4 in.	8 3-4 in.
Circumference of hand at metacarpophalangeal articulation	7 7-16 in.	7 1-2 in.
Length of index finger	3 5-8 in.	3 1-2 in.
Length of second finger	4 1-2 in.	4 in.
Length of thumb	2 5-8 in.	2 1-4 in.
Circumference of middle finger at middle part of first phalanx	2 1-4 in.	2 5-8 in.
Circumference of thigh at gluteal fold.	20 1-2 in.	20 1-4 in.
Circumference popliteal space	14 1-4 in.	14 in.
Circumference of leg at calf	13 3-8 in.	13 1-2 in.
Dorso-plantar circumference	8 1-2 in.	8 1-2 in.
Circumference of wrist	6 in.	5 7-8 in.
Circumference of ankle	7 7-8 in.	7 7-8 in.
Length from ant. sup. spinous process to int. malleolus of ankle	32 3-4 in.	33 in.

[4]Archives of Neurology and Psychopathology, Vol. 2, Nos. 2 and 3. 1899.

Case II. F. W., female, aged twenty-five years; neurotic family history. Although patient was born a healthy child at term, labor was difficult and instrumental, it being a breach presentation. At two years of age during difficult dentition she had a series of convulsions which left the right side hemiplegic. Epileptiform crises followed the palsy lesion at once. They were convulsive and confined to the right side at first; later they became general, with a right side order of invasion. Patient is feeble-minded, but able to do considerable housework. The whole right side, including the face, is hypertrophic; the bones also enter into the enlargement as shown by skiagraphs in the original publication of the case. Mild scleroderma and Raynaud's disease are also associated in this case. The following measurements and Fig. II. show the hypertrophic state of the hemiplegic parts:

	Right	Left.
Weight, 108 lbs., height 4 ft. 11 3-4 in.		
Dynamometer	46	50
Dynamometer (average) of twelve tests	42 1-2 in.	48 2-3 in.
Length from the acromion process to tip of middle finger	26 1-2 in.	26 1-2 in.
Circumference of biceps (forearm at right angles to arm)	10 1-2 in.	9 3-4 in.
Circumference below bent elbow (forearm at L)	10 in.	9 1-4 in.
Circumference at wrist	6 1-2 in.	5 3-4 in.
Circumference behind knuckles	7 1-2 in.	7 1-4 in.
Length of middle finger	4 in.	4 in.
Length of humerus (acromion to ext. condyle; arm bent at L)	11 in.	11 1-2 in.
Olecranon to styloid process (ulnar side)	9 1-4 in.	9 1-4 in.
Circumference at gluteal fold	19 1-4 in.	19 in.
Anterior superior spine to internal malleolus	31 in.	30 1-2 in.
IN RECUMBENT POSITION.		
Circumference above knee	13 1-2 in.	13 1-2 in.
Circumference of calf	13 1-2 in.	13 in.
Circumference above ankle	7 1-2 in.	7 1-2 in.
Circumference of arch of foot	8 in.	7 7-8 in.

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TREATMENT BY THE TOURNIQUET TO COUNTERACT THE VASOMOTOR SPASM OF RAYNAUD'S DISEASE.

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I have been privileged by Dr. Osler to report from his clinic the following case. The object of doing so is to call attention to a form of treatment that apparently proved beneficial in relieving a cycle of vasomotor overaction which in this particular instance had provoked a painful and protracted condition of "local asphyxia" of the four extremities.

Since Raynaud's final contribution in 1874[1] to the subject of the disease bearing his name there has been no particular advance in our knowledge of the physiological or pathological states underlying it. Its physiological basis seems to rest on the overaction in constriction of the arterioles of certain vascular territories. This overaction may be brought about by slight peripheral stimuli, which only under the abnormal conditions of the malady could produce reflexly such an exaggeration of the customary vaso-constrictor response. Thus a minimal lowering of the surface temperature for example may suffice in some cases to inaugurate an attack characterized by one type or another of the symptoms assumed by the disease. In order to account for the characteristically symmetrical nature of the vascular disturbance so often observed, Raynaud was led to believe in the existence of a lesion or state of overexcitability of the vasomotor centers themselves.[2]

Presumably the same fundamental condition, whatever it may be, is responsible—through variations in the intensity of its

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[1]Archives générales de médecine T. I., Jan., 1874. Trans. by Barlow, New Sydenham Soc., 1888.

[2]"New Researches on the Nature and Treatment of Local Aspyhxia of the Extremities," *loc. cit.* p. 182. "To sum up in a more definite form, I would say that in the present state of our knowledge local asphyxia of the extremities ought to be considered as a neurosis characterized by enormous exaggeration of the excito-motor energy of the gray parts of the spinal cord which control the vasomotor innervation."

action—for the three well known gradations in severity of symptoms: the "local syncope" (Raynaud) with exsanguinated tissues: the "local asphyxia" from venous stasis, and finally the thrombosis and gangrene, representing the terminal feature of the vascular spasm in case the latter is sufficiently severe or prolonged in its action.

The opportunities offered for a post-mortem study of the disease have been rare and the information gained has been scant and unreliable. No change in the nervous system or in the blood vessels has been demonstrated in typical and uncomplicated cases. There is no endarteritis such as occasions the somewhat similar gangrenous processes in the senile; no change demonstrable in the nerves as in cases of symmetrical gangrene associated with peripheral neuritis. Cases of chilblains, frostbite and instances of ergot poisoning with prolonged constriction of arterioles leading to anemia, asphyxia and death of the extremities possibly present points of nearest resemblance to the conditions presented in Raynaud's disease.

Forty years have passed since the appearance of Raynaud's original thesis, and yet with the exception of one or two new points in symptomatology, our understanding of the malady remains where he left it. Barlow, in his recent chapter in Allbutt's System, says: "The earliest explanation of this pathological habit appears to be Raynaud's hypothesis; namely, the vasomotor center or centers are unduly irritable, that the commonest irritant is from the periphery, for example, cold; and that the different impulses from the center lead to the paroxysmal contraction of arterioles."

The suggestion that the application of a tourniquet to an extremity, early in the course of a period of arterial spasm, might temporarily throw out of action the nerves controlling the peripheral vasomotor mechanism and thus interrupt the syncopal attack, was based purely on this theory of physiological overaction of the neuro-vascular apparatus.

The removal of a tourniquet or Esmarch bandage, which has been applied to an extremity for operative purposes, is invariably followed by a flushing of the member, peripheral to the site of constriction, with bright arterial blood. From the

embarrassment often occasioned thereby, this is a circumstance with which an operator is but too well acquainted.

This active hyperemia with increased surface temperature presumably is due to the temporary paralysis of vasomotor control to the part below the encircling bandage. Whether the pressure of the tourniquet produces a local compression paralysis or whether the resultant anemia throws the terminals of the vasomotor apparatus out of gear, it is impossible to say, but it suffices that the non-medullated fibers are "blocked" in consequence of the constriction by a more moderate and shorter application than is necessary to throw out of function those fibers conveying sensation and motion. Familiarity with the vasodilatation occurring under these circumstances naturally suggested the employment of a similar procedure as a therapeutic measure to counteract the local conditions of spasm characterizing the disease in question.

In the particular patient in whom this treatment was first instituted the symptoms were severe, the arterial spasm was pronounced and was associated with great pain. For several months a condition of local asphyxia of all the digits had been present with exacerbations of almost daily occurrence. On several occasions these attacks were so severe that slight superficial patches of terminal gangrene had affected the pads on one or more of the toes or fingers. The healing after this loss of substance had left most of the finger-tips tapering and firm. With the idea of interrupting the vaso-constrictor control over the individual digits as they became most actively involved, it was thought possible that cocainization of the lateral nerves might block the transmission of vasomotor as well as other nervous impulses and thus check the process.* This, however, was not tried as the tourniquet apparently accomplished the same purpose and controlled a wider vascular field. As is rather unusual the arterial spasm in this case extended far enough up the extremity to render the radial artery at times unpalpable at the wrist.

*It is not impossible that a subarachnoid cocainization via the lumbar meninges might serve the purpose of abruptly throwing out of action the spinal vasomotor centers in these cases.

The history of the case is as follows:

The patient, Mrs. ———, 35 years of age, entered Dr. Osler's service October 29, 1900, complaining of "blueness and pains in the extremities." Her history, taken by Dr. Cole, shows no hereditary predisposition nor associated or previous illness, malaria, lues, diabetes, etc., which might have borne some relation to her present trouble.

She was never a strong child. She had scarlet fever, two attacks of diphtheria and other infantile infections. She suffered frequently during her early years from pains in her joints, usually worse during cold or damp weather. These pains have persisted more or less throughout her life. They have never been associated with evident articular changes nor have they been severe enough to incapacitate her for her usual duties. She has suffered also since childhood from attacks of "chilblains" which never required severe cold for their incitement. From this she has suffered every winter, though with advancing years there has been no especial increase in the severity of the attacks. She says that during the attacks the toes or fingers would become "red and swollen and would itch and burn."

She was married at 24 and became the mother of two children. Following the birth of one of them she was invalided for several months as the result of a severe infection. Her catamenial history is irregular. She suffers from frequent attacks of indigestion and vomiting which seem to bear no relation to indiscretions of diet. She has been more or less of an invalid for the past six years.

Although presumably akin to her former mild winter attacks, she dates the onset of her present serious trouble from March, 1901, several months before her admission to the hospital. Shortly after an attack of la grippe she began to be troubled with burning and itching of the legs and feet. One leg she says became red and swollen though the symptoms were most pronounced in the feet and toes. An upright position aggravated the condition and recumbancy became necessary. After a week or ten days the symptoms became associated with the extreme burning pain characteristic of cases of "local asphyxia."

In a more or less aggravated form, the arms and hands also having become affected, these symptoms had persisted uninfluenced by treatment during the ensuing months. There has been recourse to morphia almost daily to obtain some degree of relief from the pain and to induce sleep. On several occasions after a particularly severe exacerbation of the condition, a small, dry, brown eschar had formed on the pads of one or more fingers or toes.

She has had no symptoms referable to the viscera, to the central nervous system nor to vision.

Beyond the condition of the extremities, her physical examination was practically negative. She was a small, well nourished woman of neurotic temperament.

Dr. Osler's note on the extremities was as follows: "The hands are slightly congested, cold and moist. There is a slow return of vascularity after compression. The terminal phalanges are blue; the finger tips especially so. The fingers are tapering and the pads show dessicating epithelium and scars of old sloughs. The nails are rigid. On the outer side of the arms are scars where the skin has been involved in the last attack.

The arteries, neither radial nor brachial are palpable. Pulse is very small and feeble.

There is a general lividity of the toes and feet. The toes show a condition similar to the fingers though more advanced. There is considerable dessication of the skin, marked blueness of the tips and scars of old healed gangrenous sloughs. They are very painful and tender to manipulation. The tip of the nose is dark colored, deeply congested, but not swollen."

Blood Examination.—R.B.C., 4,800,000; W.B.C., 10,600; hemoglobin, 65 per cent.; coagulation time, one minute, twenty seconds.

Urine Examination showed nothing noteworthy. Almost daily observations over a period of two months showed no trace of hemoglobin.

With no evident amelioration, the patient's symptoms continued practically unaltered for the following two weeks. There was more or less constant discomfort with attacks of severe burning pain once or twice daily associated with blueness of the extremities. These exacerbations were only influenced by morphia. Although galvanism was not employed, the usual forms of treatment seemed to do little more than temporarily palliate the severity of the symptoms.

On November 10, during an exacerbation of pain and asphyxia chiefly affecting the left upper extremity, the flat rubber bandage was first employed as a tourniquet about the upper arm. Its application occasioned considerable local discomfort and pain referred to the side of the thorax (intercosto-humeral?). On its removal after one or two minutes' application a bright flush of the extremity followed with increase of surface temperature and a much more readily palpable radial artery. The vasomotor relaxation lasted only a short time, but the patient expressed a sensation of considerable relief. From this time seems to date the beginning of her improvement. The

tourniquet was applied daily to one or another member according as the symptoms indicated. The local discomfort which it occasioned at first was subsequently less severe or was better endured and the application finally was extended to periods of five minutes or longer. The relief to the burning pain in the fingers was so pronounced that after the first few trials the patient was not only very willing to submit to the temporary discomfort of constriction, but would call for it.

An attempt was made to apply the tourniquet as early as possible in the period of exacerbation of pain and lividity. These periods occurred, however, with such irregularity that it was difficult or impossible to anticipate and ward off the vascular spasm by applying the tourniquet as a prophylactic measure, although an endeavor was made to do so. Improvement in the circulatory condition, however, was noticed almost from the first. The patient soon ceased to require morphia to induce sleep and the attacks became less and less frequent and of decreasing severity. After two or three weeks she was able to get up and move about her room without discomfort. Exposure from cold as from driving in the open air brought on no further trouble. She left the hospital apparently quite well on December 28, having gained ten pounds in weight, and having had for the preceding two weeks no recurrence of her distressing symptoms. The extremities at this time had resumed their original physiological tint.

During the patient's residence in the hospital while the recurring spasms with pain and lividity of the extremities were still severe, careful observations on the blood pressure were made by Drs. Cole and Erlanger. They failed, however, to demonstrate any characteristic rise in the general arterial tension other than that which could be accounted for by pain. It is probable that the vascular territories subjected to the vasoconstrictor action at any one time were too circumscribed to occasion a rise in the general blood pressure of any significance. A corresponding constriction of the splanchnic field would have produced a tremendous rise in arterial tension. A "local asphyxia" of the splanchnic venous system on the other hand, corresponding to the degree of stasis in the extremities, would have brought about a great fall in blood pressure. Apparently in this case, as heretofore stated, there were no circulatory disturbances referable to the viscera or central nervous system.

For the remainder of the winter months, following her discharge from the hospital, the patient remained free from symptoms and was able to resume her household duties with enjoyment. On one occasion in the spring, after fatigue and expos-

ure to cold, she was threatened with a recurrence of the trouble. Once more her hands became livid in color and so painful that her attending physician administered morphia, but without giving her any relief. Recourse was made once more to the tourniquet with immediate improvement in the condition, and after a few applications the dreaded symptoms entirely disappeared. Since then up to the present writing she has had no return of trouble.

Galvanism in one form or another has proved of most benefit in the treatment of these conditions. This form of therapy is strongly advocated by Raynaud in his second essay, in which striking examples of improvement under its employment are cited. The rationale of the treatment depends upon the supposed relaxation of the overacting vasomotor centers in the cord consequent upon the passage of the electrical current.

The treatment by the tourniquet is most simple of application and from its apparent success in this instance may be possibly deserving of further trial. It depends seemingly in principle upon the physiological "blocking" effect of the elastic constriction on the peripheral vasomotor nerves. This temporary interruption of function allows the terminal arterioles to completely relax and a state of arterial hyperemia to take the place of the local asphyxia which has resulted from the pre-existing spasm. If employed early enough or repeated with sufficient frequency, as this case suggests, the hyper-excitability of the center, if that is the underlying cause of the spasm, either subsides or else, and what is more probable, the local vascular conditions in the extremities become so altered by the periods of active circulation that slight peripheral stimuli no longer provoke the intense reflex constrictor responses.

The treatment, of course, has its limitations, and is not applicable to a case where vascular spasm affects territories other than those confined to the limbs.

THE HISTORY OF THREE CASES OF PECULIAR MOTOR MANIFESTATIONS IN THE INSANE.[1]

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These cases are reported as illustrating the bizarre and extreme manifestations of motor disturbance as they occur among the insane. They practically never have an organic basis; and although they frequently seem to have a focal origin; yet, even when recovery does not take place, and the patient dies, we have, so far, never found any evidence of a focal lesion.

Our experience with the motor disturbances associated with insanity, and especially those degenerative forms: disseminated cerebro-spinal sclerosis, the ascending form of general paralysis, and hereditary chorea: which link disease of the brain with that of the general nervous system; suggests the possibility that nervous disease independent of insanity may be due to like causes, operating in a more limited field, and resulting from a more definite reduction of potentiality, or exhaustion of capacity. In other words; that the diseases of the bulbo-spinal nervous system, are not so much specific conditions, as they are the sequence of slowly or rapidly developed degenerative change in certain parts of a congenitally defective structure, in which the capacity in these parts was limited; so that a sufficient strain, overuse, or disease, would set up a process of decay which would be progressive. It is interesting to note in this connection that we not infrequently see, among the insane, almost every form of focal disease imitated by the effect of chronic uremic poisoning upon the general nervous system; and some of these effects persist during the life of the patient.

G. Z., German, married, a farm hand, 43 years old, 5 feet 10 inches high, eyes blue, and hair brown. Had recently come to America, and no history of his past life or heredity could be

[1]Read at annual meeting of the American Neurological Association. June 5, 6 and 7, 1902.

obtained. It was stated, however, that he was "partly insane" when he came to America. He was brought to the hospital because he had persecutory ideas, feared personal injury; and was disposed to be violent toward his family and neighbors.

He was in good physicial health apparently, had a good appetite, and slept well. He appeared to be intelligent, had good manners, spoke fluently in his own language, and gave evidence of a fair degree of education. At first he was very much disturbed, suspicious of every one, and in constant fear of personal injury. He was either dull and indifferent, or disturbed and agitated; and when in the latter condition complained of constant dull headache, and said that he did not sleep well. However, he took part in the regular occupations of the ward, and also went out walking. After he had been in the hospital for about two months, during a period of unusual depression and agitation, he ran away; but was found the next day hiding in an old farm house. He said he feared he was to be "killed;" begged that he might be "shot and not tortured;" also that his "sentence" might be "commuted to life imprisonment." The headache was worse at this time, and he developed an angioneurotic edema in the left forearm. His physical condition exaggerated his morbid self-consciousness and depreciatory ideas; so that everything in his surroundings was pregnant with suggestion. He was too confused to recognize any well-defined expressions of hostility from those about him, although there was evident auditory hallucination; which, associated with some untoward experience in his past life, suggested the persecutory ideas which dominated his active consciousness and filled him with dread and fear. During this time, however, he took plenty of food, and when he was at work was apparently comfortable. He did not sleep well, however, and grew more depressed; careless and indifferent with regard to his clothing and appearance, and took no interest in his surroundings. During the next month he took on flesh very rapidly, became more stupid and indifferent, his face expressionless, and he did not answer when spoken to; although he apparently understood what was said to him. It was noted at this time that there was some paresis of the muscles of the left forearm, and general paresis; in so far as to make him awkward in his movements, so that he stumbled in walking and was unable to work. In the early part of the next month he began to complain of being unable to see well with his right eye. One week later he could not see well with either eye, and could not read. Shortly after this he was found, soon after going to bed, to be in a tonic convulsion, apparently involving all of the voluntary muscles. He remained rigid for more than an hour, was

completely anesthetic, but apparently fully conscious. The next morning there was marked evidence of paresis on the left side, including the face. He could move about, but was very awkward. The motor symptoms disappeared gradually during the next month, but he had occasional convulsive seizures, which were always tonic. He lost in weight and strength, took little food, and was apparently neither able to work nor read.

Up to this time he had always conversed intelligently on subjects with which he was familiar, but now he became incoherent. He was kept in bed on account of his physical condition, and because he complained of pains in various parts of the body. There was no tenderness nor soreness, however; and during the day, when not watched, he would get out of bed, stand in a corner of the room, and watch furtively, as if he feared to be attacked.

There was no marked variation in his mental condition during the next month, and his persecutory ideas persisted. Toward the latter part of the month he had another convulsion lasting several hours, and the muscles of the jaw remained rigid for four days. During this convulsion, and afterward, he was apparently insensible; respiration became very rapid, the rate reaching as high as 60. During this time only the left side of the chest expanded. There was no cyanosis. He slowly recovered his former physical condition, but still complained of various pains, especially under the eyes and in the right ear.

During the next month he had an attack of dysentery, which was epidemic in the house at the time. His illness did not affect his mental condition, but there was no return of the convulsions.

During the next two months he had occasional convulsive seizures of short duration, and gradually passed into a condition of painful stupor. The vegetative functions were performed normally, but the excretions were passed apparently unconsciously. During the latter part of the period the convulsions became irregular in their onset, and instead of simple rigidity, the body was rotated to the left, and the knees drawn up to the chin. He had at one time 20 successive convulsions within a few hours. Even when the muscles were apparently relaxed there was marked resistence to any effort to flex or extend the limbs; the condition resembling pseudo-catalepsy.

During the next month the convulsions were comparatively frequent, and of such a character as to warrant detailed description. The patient was in bed, apparently unconscious of his surroundings. The body would suddenly become rigid; respiration hurried and noisy. After a varying interval, the eyelids would begin to quiver; then the distal phalanges of the fingers and

toes would begin to move; and this movement progressed symmetrically and successively from joint to joint, until all four limbs were in motion. Then the muscles of the trunk became involved from below upward, and finally the muscles of the face. The movement was that of a violent tremor, apparently affecting all of the voluntary muscles. It subsided gradually until the tremor became fibrillary. Then there was complete relaxation. During the stage of rigidity he could be placed with the back of his neck resting on one support, his heels on another, like a trestle board; and considerable weights could be piled on his abdomen without affecting the rigidity.

During the next year there was very little variation in his condition. He remained in bed, was stupid and filthy, but took plenty of nourishment, and slept well. He also had occasional convulsions. In the latter part of the period he began to cough and expectorate. Soon after this he began to be disturbed, got out of bed, and if not watched would attack the other patients, and break windows. One day he beckoned the physician to him and told him that he had been advised to speak, and to say that he had a pain in his breast. From this time on he spoke freely and intelligently, and began to take an active interest in his surroundings.

He was weak physically, and during the next two months coughed and expectorated a great deal. His ankles became edematous also. So far as could be determined, the condition of the lungs was due to a chronic edema. There was no elevation of temperature, and no tubercle bacilli were present. By midsummer he began to improve physically, and from this time on improved steadily, both mentally and physically; until after a period of four months he was entirely well and went home. Nothing has been heard from him since.

J. B. S., German, single, 40 years old; eyes gray, hair brown; height 5 feet 8 inches, weight 147 lbs.; laborer.

Nothing could be learned of his family or personal history, except that his parents were living, in good health; but the father intemperate. The patient is said to have always been "feeble-minded," and since he reached manhood an habitual drunkard. Ten days before admission he was struck on the head with a rock. A week afterward he became disturbed, wandered about trying to hide himself; said that he feared that he would be murdered; that he saw people after him, and heard them threaten to kill him. He also complained of pain in the head and flashes of light before his eyes.

He was too much disturbed to admit of a careful physical examination. He had a good appetite, but digestion was im-

paired, and he was constipated. The urine was decreased in amount; sp. gr. 1.025; urea and chlorides reduced, but no other abnormality was apparent. The eyes were suffused, conjunctivæ congested; expression that of furtive suspicion. He was disturbed, apprehensive; sat up in bed, swaying from side to side, wringing his hands, and beating his breast. When any one came near him, he would shake his head violently, whine and snarl; shout, "Murderers, murderers, murderers, all murderers; they are going to murder me; the rich devils are after the poor man." When alone he was quiet, and with one to whom he was accustomed he would talk rationally about ordinary things. During the first forty-eight hours after his admission to the hospital he was more quiet, took nourishment freely, and slept fairly well at night. The constipation was relieved, but the urine continued scanty in amount. Any attention disturbed him. He would growl like a dog, strike himself in the face; and attempt to bite and scratch the nurse. During the periods of quiet he would say that he could not help acting as he did, because people were after him, trying to kill him. During the next week there was no material change in his condition. He slept well under the influence of hypnotics, took nourishment when given to him, and his bowels moved daily under the influence of laxatives. In the day time, however, the visual and auditory hallucinations were persistent, and the fear of personal injury ever present. There was no elevation of temperature, although the surface of the body was flushed, and the skin hot and moist. During the next two months the only change in his mental condition was a growing incoherence. He did not take food voluntarily, and became filthy in his habits. At the end of this time he became more restless; his former loud denunciation lapsed into incoherent muttering, and he was constantly trying to get out of bed. When allowed to do so he would crawl under the bed and try to hide himself. During the next month he grew weaker, did not try to sit up, and when put on his feet apparently could not stand. When not disturbed he would lie with his eyes closed, muttering and whispering incoherently to himself.

During the next year his condition remained practically unchanged. He had to be fed mechanically, was filthy in his habits, the delirium persisted; and the paresis of the lower extremities became more and more marked. His general physical condition was better, however. A little later he began to complain of loss of vision, and could not distinguish the different objects about him; but said that he could see "squirrels," "crickets," "snakes," and "bugs," about the room. During the next three months he was quite stupid, filthy in his habits, and

most of the time had to be fed mechanically. The loss of vision was apparently complete, and there was not even a light reflex. He was occasionally disturbed for short periods; usually manifested alarm for his personal safety; complained of many ill feelings; and also of disagreeable sights and sounds about him. By dint of persistent effort, and questioning, however, he could always be made to manifest some intelligent appreciation of his immediate environment.

About this time there began contracture of the muscles of the leg and thigh, and during the next month the legs became flexed upon the thighs, and the thighs partially flexed upon the abdomen. They could not be extended, and he always complained of great pain when the effort was made to extend them. During the next two months he improved physically, but still had to be fed mechanically. As he grew stronger he became boisterous; complained bitterly of the way he was abused, and was constantly talking about the things he saw and heard about him, which were alarming or offensive. It was apparent at this time, also, that he could see something of the objects by which he was surrounded; although he would disclaim this capacity if asked about it. During the next nine months he gradually improved physically; his body became robust and vigorous, but the lower extremities remained rigidly contracted. The muscles were extremely atrophied, and there was very little evidence of sensation. He was noisy, garrulous; alternately fawning in manner and fulsome in speech; or profane and abusive. He also became very self-conscious, enjoyed personal attention, and resented any apparent neglect. Visual and auditory hallucinations were active and persistent, and when his attention was not attracted by some one speaking to him, he was constantly and vociferously answering what he heard or disclaiming against what was said. He was especially troubled because the voices made lewd proposals to him, or accusations, and these he attributed to the nurses. He insisted that he could not see, but recognized people by their voices. He would not eat in the presence of others; but if food was put where he could get it, and he thought no one was near, he would eat it.

Sept. 1, 1899, he was put in bed, complaining of colic and diarrhea. There developed an entero-colitis, with frequent bloody stools, and tenesmus. He would not take food voluntarily, slept little; complained a great deal, but would not answer questions. He became very much emaciated, and very weak. After the first week of his illness, his legs gradually straightened out, the flexion of the thighs relaxed, and he could move his lower extremities with comparative freedom.

His illness lasted three weeks, and at the end of this time

he was almost a skeleton; but with the beginning of convalescence his appetite became ravenous, and he gained rapidly in weight and strength. He insisted, however, that he could not walk; although he dressed and undressed himself, and made his own toilet. As he grew stronger, the visual and auditory hallucination returned; he heard and saw "Satan," and numerous "devils" about him; often became noisily profane and obscene; denouncing them for tormenting him. He walked about with crutches; and it was noticed that, when he thought he was not observed, he would drag the crutches and walk without their aid. In the spring he was carried out of doors; where he could crawl about, talking to himself and complaining to any one who would listen about his ill treatment by "Satan" and the "devils." One day, when he thought he was not observed, he got up from the grass and walked quite a distance, but as quick as he heard any one near he would drop to the ground. The next day he was told that he must walk to the house himself, as no one would carry him in. He remained out for an hour after the other patients came into the house, but when he heard them eating supper could not stand it any longer; got up from the ground and came hobbling into the house, whining and complaining about his ill treatment. After this he made no pretence of being unable to walk nor of not seeing; rapidly regained his strength, became robust, and spent most of his time in walking.

He is now very vigorous, spends all of his time out of doors; his vision is acute, but the eyes have the expression of sightlessness. There is no evidence of motor involvement of any kind, but he is demented, although constantly plagued by the devils he sees and hears; the voices often coming from inanimate objects about him, and accusing him of all sorts of misbehavior.

A. F., a girl, 14 years old. She was quite stupid when admitted to the hospital, but in good physical health. No history of the family was obtained, nor of the patient, except that she was said to have suffered from convulsions for about a year before coming to the hospital, and had been quite violent at times. She was evidently a low-grade imbecile. She would answer questions, express her wants and care for herself; but was restless, irritable, and there was occasional muscular twitching; but no convulsions after coming to the hospital. The twitching gradually gave way to incoördination; she ceased to be able to walk, and was put in bed. The degeneration proceeded rapidly, and paresis became quite marked. The vegetative functions remained intact, and those of the bowels and bladder were performed automatically. For a time she was emaciated, but

afterward became well nourished. Her muscular activity was automatic, and the movements of the limbs were purposeless like those of an infant. Her mental functioning was entirely rudimentary; she laughed and cried like a baby, but had no articulate expression; was entirely without comprehension of spoken words, and indeed an infant; without capacity to relate or coördinate external impressions.

After a time she began to make articulate sounds, and gradually the simplest coördinate movements; such as grasping an object, and carrying it to her mouth. Next she began to sit up, then gradually to get about, and finally to walk. After getting up and about the ward, a process of reëducation was begun, and she eventually acquired sufficient capacity to care for herself, sew, read a little, and carry on ordinary conversation on subjects pertaining to her environment.

It will be noted that in the first and third cases there was no paralysis, but on the contrary, so far as could be determined, muscular power remained intact. In the first case the apparent loss of power was due to general spasm, while in the last it was due to incoördination, and it is probable that the inability to articulate was also the result of incoördination.

The nearest approach we have to a similar condition due to organic involvement is in those cases of chronic hypdrocephalus where the fluid in the lateral ventricles accumulates so rapidly that the cortex is compressed against the cranial wall, and there results general spastic tremor and sometimes convulsions. Here of course the source of the motor manifestation is in the mechanical irritation from pressure. Another analogous condition is that existing in status epilepticus, but here too the cause is practically mechanical, the condition resulting from engorgement and edema, which sometimes have their origin in failure of the function of the kidneys, but more often in failure in the function of the brain, resulting from exhaustion; and, post-mortem, the evidence of cell fatigue is all that is found.

The third case was incorporated in this record because it offers the best basis for an hypothesis as to the pathology of these conditions; and unfortunately any explanation must be hypothetical, because we have no histologic basis from which to determine their pathology.

The complete recovery so far as motor capacity was con-

cerned, the return of the power to articulate, the later improvement in verbal capacity as the result of training, and the acquisition of a fair degree of manual dexterity, show that no permanent damage was done to the neurone. We have not had many opportunities for study of the brains of the feeble-minded before the ordinary terminal processes of degeneration had begun, but the few cases we have examined did not present anything peculiar histologically, outside of the gross differences common to the defective. There was evident only an apparent numerical difference in the cells in a given cortical area, with a relatively greater proportion of neurogliar tissue.

The most apparent explanation is the general reduction of capacity from exhaustion or autointoxication, resulting in the production of excessive irritability and hypertension. This explanation might apply to the first and second case, but not to the third, for the patient was in good physical condition at the beginning of the manifestation of motor disturbance.

However, the progressive emaciation which went along with the loss of muscular coördination would suggest that there was some trophic involvement. In the record of a case of general paralysis reported by me ("Sensory and Sensory Motor Disturbances," JOURNAL OF NERVOUS AND MENTAL DISEASE, for Oct., 1892) this same progressive development of incoördination with loss of the power of articulation was present, and in this case too there was a relatively complete restoration.

To the careful student of insanity there is no one element so conspicuous as the tendency of its manifestations to occur in cycles. Excitement to be followed by depression in both motor and mental disturbance, or its converse; with a period of apparent restoration between. Furthermore this wave-like tendency is most marked in those who are most defective, and is most conspicuous in those individuals who break down in early life.

In the defective individual whatever gives rise to the outbreak of mental disturbance also starts the process of reduction in capacity for coördination of functional activity in the nervous system, and this process once started seems to have a tendency to proceed definitely to a certain degree, and this is most

marked in those in whom the process of degeneration starts earliest, and in these subjects the reduction is most extreme. The presence of motor manifestations in some, and not in others, is accounted for by the antecedence of some form of motor involvement, as in the cases included in this report. In two of which there was hysteria, and in the other convulsions.

Each cortical cell has a definite potentiality, and a capacity governed by the completeness of its reconstitution. Temporary conditions may exist, which exhaust capacity, and confusion supervenes; to be followed by incoördination. This is illustrated in the mental and motor disturbance accompanying typhoid fever, pneumonia, or septicemia; and in more marked degree in chronic uremia. These conditions are recovered from, however, and capacity is restored. But in the defective individual, the potentiality of the neurone is more limited, and its capacity for reconstitution is lessened; so that there is a progressive failure of power; first manifested by increased irritability, then by spasm, incoördination, and finally by exhaustion. If the vegetative functions remain intact, there is a gradual recuperation, with restoration of capacity and return of function. We can only presume that the changes which produce these results are chemical primarily, and have their origin in instability which is extreme, with the resulting tendency to respond excessively to slight stimuli.

A CASE OF UNUSUAL CEREBRAL HEMORRHAGE.

By O. T. Osborne, M.A., M.D.,
PROFESSOR OF MATERIA MEDICA AND THERAPEUTICS AT YALE UNIVERSITY.

Mr. X., a student, aged twenty-four years, first came under my care on November 19, 1900. I was sent for hurriedly to find him in a half-stupor with projectile vomiting, almost pulseless at the wrist, but covered with a warm perspiration. He had been swimming in a tank, but had been able to dress himself before the attack culminated.

Examination of the urine showed it to be milky in character on account of the presence of phosphates, with no other abnormal condition. The patient received a hypodermic of strychnine, and a nurse was sent to watch him. During the next few hours some improvement in his condition occurred. The symptoms of the next four days were those of pressure on the medulla oblongata with marked Cheyne-Stokes respiration, the latter lasting four days. The temperature was subnormal, down as low as 97°; the pulse was sometimes as low as 48. There was stiffening of the back of the neck and occasional projectile vomiting.

A history of a similar attack, although not as severe, about three weeks before, was obtained. At that time he had had some jaundice, and had been noticed to yawn a great deal, this showing disturbed respiratory function, but none of the distinct symptoms of implication of the medulla oblongata were at this previous period present.

A diagnosis was made of pressure of that region of the medulla oblongata which contains the pneumogastric and spinal accessory centers. This would account for the reflex vomiting, slowing of the pulse, and stiffening of the sterno-cleido-mastoid and platysma muscles, and also the disturbance of the respiratory center, unless respiration is a pneumogastric function.

During the attack in November he could be aroused, but was half stupified for four or five days. He could not at first

be raised or moved, or even take a drink without vomiting. Gradually the vomiting ceased, the respiration improved, the stiffening of the back of the neck lessened, the temperature came up to normal, and the pulse increased in frequency. There were no cerebral symptoms at this time except headache, which was not very intense or very severe, and was largely basal. The diagnosis of a gumma or glioma was made, and mercurial inunctions were begun and potassium iodide was given by the mouth.

In three or four days when he could be properly aroused and his answers were intelligent, a history of an initial lesion three years before was elicited, for which treatment had been given for three months. There had never been any secondary symptoms.

The patient gradually improved, and on November 29 he was sitting up, and in a few days more was down stairs. On the first exercise, he had slight fainting attacks with rapid pulse, running up sometimes to 110 and 120, and a little increase in temperature; evidently purely functional disturbances. The signs at this time showed distinct absorption of whatever had been causing pressure on the medulla oblongata. He gradually got out-of-doors, and on the 14th of December, 1900, came to my office with a history of constant improvement. On the 18th of December he went skating, and soon after behaved queerly, had epileptic attacks, screamed, was delirious, then stupified, and then would again be aroused and complained of intense headache, but without any symptoms of pressure on the medulla oblongata. On the 19th he became difficult to manage, and required two attendants. He was then taken to the hospital with the symptoms of cerebral pressure, and soon showed some fever, and an irregular and rapid pulse. Some retinitis was now present, and a peculiar intermittent facial paralysis of the left side developed. His condition became temporarily better only to again grow worse with finally stupor ending in death in about ten days.

Professor H. B. Ferris made the autopsy which showed a friable disorganized mass about the size of an English walnut in the anterior part of the right cerebrum, with a clot hanging

to it which had made quite a space for itself in the brain substance. This clot was continuous with a large clot, apparently of recent origin, in the lateral ventricle on the right side. Another clot of earlier formation was in the third ventricle, and a third clot, I thought still older in appearance, was in the fourth ventricle and pressed on the medulla oblongata.

Microscopical examination of the above mass showed it to be an old blood clot. The arteries of the brain were everywhere healthy.

NEW YORK NEUROLOGICAL SOCIETY.

May 6, 1902.

The President, Dr. Joseph Collins, in the chair.

A Case of Primary Myopathy.—Dr. J. Ramsay Hunt presented a girl of sixteen having a negative family history. One year and a half previously there was noticed a difficulty in pronouncing words, and also a nasal twang. A few months later there was some regurgitation through the nose, and it was noted that she did not close the eyes completely during sleep. Examination showed marked deficiency of the muscles of the face on voluntary and emotional innervation. The orbicularis oris and the orbicularis palpebrarum and the zygomatici were chiefly affected. The palatal muscles were motionless. The muscles of the upper arm and shoulder were small. The tendon reflexes were active, but there was no evidence of involvement of the pyramidal tract. The speech mechanism was easily fatigued, articulation becoming less distinct on prolonged effort.

Tumor of the Brain.—Dr. Max Mailhouse, of New Haven, presented a brain taken from a woman who had been admitted to the New Haven Hospital on March 13 with a history of severe headaches for a year and a half, and considerable mental disturbance. Speech was incoherent and muttering. There were exaggerated knee-jerks; also ankle-clonus and increased plantar reflexes. The optic nerves were atrophied and she could only distinguish light. Twitching of the muscles of the eyebrows was observed the first night, and she was so restless as to require restraint. During the six weeks she was in the hospital there were two or three attacks of vomiting. She complained constantly of headache and thirst. On April 14, when examined by Dr. Mailhouse, there was slight strabismus from weakness of the left internal rectus; the knee-jerks were exaggerated and there was ankle-clonus on each side. There was loss of vision. Her mental condition was fairly good. She became more restless and talkative and the pulse became more rapid and irregular, and two days before death she became unconscious, and died in this condition. The autopsy was made on the day after death. It revealed a tumor in the interpeduncular space, and examination of the tumor showed that the growth either originated in or soon involved the optic chiasm. The tumor pressed upon the left third nerve, and also upon the facial tracts above the pons, causing the spasmodic twitchings of the facial muscles. No report of the microscopical examination had yet been received.

Paralysis of the Serratus Magnus.—Drs. Joseph Collins and I. Abrahamson presented a woman of twenty-eight who had been in fair health up to last November. At this time she had had an attack of suppurative tonsillitis, which was followed by suppuration of the glands at the posterior border of the sternomastoid muscle. These glands were incised and much pus evacuated. About seven weeks afterward the shoulder was noticed to project upward and it was the seat of a throbbing pain. When seen the left scapula presented a wing-like appearance as a result of what appeared to be an isolated paralysis of the serratus magnus muscle. Some of the fibers of the supraspinatus and of the infraspinatus showed slight reaction to the faradic current and

some slight atrophy. There was a large linear post-operative scar along the upper posterior border of the sterno-mastoid muscle. There was some enlargement of the glands on the left side of the neck. The diagnosis was based upon the position of the scapula while at rest, on the peculiar limitation of movement when the arm was brought forward at a right-angle, on the absence of sensory disturbances and of symptoms pointing to spinal origin, and on the presence of degeneration reaction. The lesion was evidently a post-infectious neuritis involving the posterior thoracic nerve, probably the portion in the body of the scalenus. The chief features of interest were: its peculiar etiology; its occurrence in a female; the absence of trauma and of any of the ordinary causes; its late recognition; the change in the position of the scapula when at rest; the presence of scoliosis as had been observed by Stromeyer in a few cases.

Dr. W. M. Leszynsky said that most of these cases of serratus magnus paralysis were due to pressure or to progressive dystrophy, but he had shown a case to this Society in which it had followed an infection—a pneumonia.

Atypical Syringomyelia.—Dr. Joseph Collins showed a case which he had diagnosticated as syringomyelia. The patient was a stationary engineer, fifty-seven years old, who had lived a temperate and healthy life, not exposed to syphilitic or inorganic poisons. Three years ago he noticed, on going down stairs, that he tripped easily, and that he was getting clumsy in the use of his feet. About the same time he remarked that the little and ring fingers of both hands were becoming stiff, as he termed it. He found difficulty in using these fingers dexterously, and in appreciating objects coming in contact with them. Soon these fingers became deformed; a flexor deformity of the second and third phalanges was noticed, which was gradually progressive and which was dependent upon atrophy of the hypothenar muscles and the last interossei. At the same time the loss of sensibility in the ulnar distribution progressed. About a year ago a slight sore developed on the ball of the right foot, and had become a typical perforating ulcer. The patient presented now the following condition: (1) atrophy of the hypothenar and last interossei muscles, and consequent deformity; (2) glossiness of the skin of the hands; (3) analgesia and thermo-anesthesia, and partial tactile anesthesia in the little fingers and the ulnar half of the ring finger; (4) thickening of the ulnar nerves, rendering them palpable; (5) increase of mechanical irritability of the muscles of the upper extremities; (6) typical perforating ulcer in the ball of the right foot; (7) analgesia and thermo-anesthesia of the toes and the outer half of both feet; (8) slight exaggeration of the tendon jerks in the lower extremity; (9) no deformity of the spine and no symptoms referable to the cranial nerves; (10) no disturbance of the sphincters or of the sexual function. Dr. Collins said that the case was not a typical one of syringomyelia, but that diagnosis seemed more likely than any other. The condition was a progressive one.

A Case of Raynaud's Disease.—Dr. B. Onuf presented a young man with Raynaud's disease that had existed for about two years. Sensation was normal during the attacks. There was a history of rheumatism of the joints. He was a sailmaker by occupation, and in his work the tool used caused pressure on the right palm. The urine showed an abundant deposit of urates. He had not been improved by any method of treatment so far employed.

Mental Dissociation in Depressive Delusional States.—Dr. Ira Van Giesen was the author of this paper, which, he said, represented some

of the work formerly done in the New York State Pathological Institute. The subject of the report was a Russian of twenty-six having a good family history. In February, 1900, he had begun to suffer from insomnia, headaches and loss of appetite, and had become despondent. The general health had rapidly deteriorated, and a local physician, on examining him, stated that his trouble was largely indigestion, and that there were "lumps" in the bowel. This was the foundation of a delusion which the patient fondly cherished. There was also a belief that there were worms in the intestine which worked upon the lumps and broke them up into minor lumps and sent the latter throughout the body. The patient believed that he was rescued from his dire distress by three agencies, viz., the spleen, the soul, and the veins, the soul being the scavenger and the spleen the director. When the attention of the soul was distracted this work was not well done. As might be expected the patient was extremely depressed and his physical condition suffered. He could only speak readily on the all-pervading subject of his delusion. The loss of attention was marked except as regards the systematized delusion. No gross motor disturbances were present. The patient reacted slowly to external stimuli. There were no sensory disturbances, and no hallucinations, and no tendency to self-destruction. He could fully realize his environment and his relations to space and time. As soon as he passed into hypnotic trance a metamorphosis occurred, the patient passing from intense depression to a state of great exaltation. Despite this the focal delusion persisted and appeared to be far better organized. This clearly pointed to the fact that either the state of depression was one of secondary formation or the delusion, being secondary in its origin, had gained sufficient strength to stand by itself, even after the emotional basis had been withdrawn. The latter alternative seemed to be the more probable. In his trance he could vividly remember all that had taken place in his waking life, and, on the other hand, he could recall fairly well what happened while in the trance. Later on the patient at one time passed into a deeper trance and then passed from a state of inexpressible delight to one of grave composure. In the last trance personality he could remember all the experiences of the other trance personality and of the waking personality. The relation of these three personalities was described diagrammatically by three concentric rings. The central one was the melancholic personality, and outside of this were successively the one ring representing the first and second trance personalities. Of the three personalities, the waking was pathological. The first trance personality was an exaltant, while the second trance personality approached closely to the patient's healthy condition. The course of these personalities resembled quite closely what was observed in circular insanity. These alternating personalities were, however, ephemeral. Soon the first personality shrunk and finally disappeared altogether. In the course of time the first trance personality disappeared and never returned. The mood of the second trance personality then lost some of its former seriousness. The tendency was for the intermediate personalities to disappear and the last one to become the dominant one. The process of evolution of species in general was one great illustration of the process just described. Throughout all of these transformations the central delusion remained unshaken. The great assimilating power of this delusion was wonderful. Various suggestions were given to this patient; although they were designed to break up the nucleus of the delusion they were turned about by the patient and fed into this systematized delusion. It was necessary to follow the patient in his delusion and play into the hands of the latter in order to make the suggestion take root. Direct suggestion during deep hypnosis was the

usual method of breaking such a strong delusion, but the objection to this method was that it was apt to be only temporary in its effect. The method of emotional substitution was especially valuable in a case of this kind. Some unimportant sensory changes were first attempted, as for example, the abolition of the unpleasant thermal changes in the "lumps," but they were only partially successful. The effort was then made to fuse the different personalities. During hypnosis dreams were suggested to the patient, with the object of effecting changes in the central delusion. For example, in one of these dreams his father told him that these lumps would go away. These dreams impressed him deeply though slowly. When the good dreams had become dominant the spleen and soul began to drop out, and then the galvanic current was substituted for the soul with great benefit. Small spots were next substituted for the large lumps. The spots were gradually confined to certain definite areas instead of being vaguely disseminated. The patient's melancholia finally disappeared completely and he had now resumed his original vocation. The case was regarded as a triumph for this mode of treatment, and as a type of many other cases now in the State Hospitals for the Insane.

Dr. Mary Putnam Jacobi asked whether the transition of the two personalities had occurred spontaneously or had been brought about by some special maneuver.

Dr. Henry Rafel asked if there were any other case on record in which hypochondriasis had been cured in this manner by hypnosis.

Dr. Van Giesen said that the paper had been prepared by his former associates at the State Pathological Institute. The transition of the personalities had occurred spontaneously. He was not sufficiently familiar with the literature to answer positively the second question. If hypnosis had been used in the ordinary way the result would have been only transient; a permanent cure had been effected by a thorough understanding of the whole mechanism of hypnosis. This patient had been cured eighteen months ago.

Periscope.

Archiv f. kriminal Anthropologie und Kriminalistik.

(1902. Vol. 9, Nos. 2 and 3.)

6. Contribution from Legal Medicine concerning the Use of Secret Codes among Criminals. SCHUTZE.
7. Study of Evidences, especially Foot Traces. SCHUTZE.
8. The Main Advances in Criminal Anthropology in 1901. NÄCKE.
9. Internal Stigmata of Degeneration. NÄCKE.
10. How do Homosexuals know One Another. MOLL.
11. The Case of Fischer. SIEFERT.
12. Criminal or Insane. POLLACK.
13. Value of Evidence. LELEWER.

6. *Concerning the Use of Secret Codes among Criminals.*—The author discusses several codes used by criminals in writing to each other; these were in part explained by the criminals, in part deciphered. The codes are based on various principles, but are all in principle very simple. There are, in the first place, the ordinary arbitrary codes, in which each letter, or at times combinations of letters, is represented by some other accepted literal equivalent. Other codes are built on a figure basis, *e.g.*,

a|b|c
d|e|f
g|h|i

the tit-tat-toe code, and so on, "a" being represented by $-^{|}$, "j", the first letter of the second series by $\lrcorner$, "s", the first of the third by $\lrcorner\!\!\cdot$, etc. Finally, there are codes in which a single word, the component letters of which are numbered, serves as a key to the system. [The tit-tat-toe code is widely used in America.—J.]

7. *Study of Evidence, especially Foot-traces.*—A narrative article.

8. *Advances in Criminal Anthropology.*—A review for 1901.

9. *Internal Stigmata of Degeneration.*—From a study of 104 paralytics and a comparison of their organs with those of 108 average bodies from the public hospital in Chemnitz, the author selected the following anomalies as fairly characteristic of the former group. Lungs: anomalies of size and lobation. Heart: diminution in size, also of vessels; double apex; potency of foramen ovale or ductus Botalli. Liver: anomalies of size and form, multilobar type, multiple and deep fissures, absence of Spigelian or quadrate. Spleen: anomalies of size and shape, lienculi, lobation. Kidney: anomalies of size and shape, asymmetry, horse-shoe kidney, double pelvis or ureter.

10. *How do Homosexuals recognize each Other.*—In addition to the signs ordinarily made use of by one individual to attract the attention of another, the homosexual is known to his fellow by over-ornamentation in dress—rings, powder, etc. In addition, there are certain arbitrary signals, *e.g.*, the wearing of different colored carnations. Finally, a characteristic motion of the tongue is described.

11. *The Case of Fisher.*—An analysis of a *cause cèlébre* and of the criminal agent.

12. *Criminal or Insane?*—The case of a degenerate, semi-imbecile, and alcoholic, a wanderer between prison and asylum.

13. *Value of Evidence*—especially that of children. An essay.

R. WEIL (New York).

Archivio di psichiatria, scienze penali ed antropologia criminale.
(1902. Vol. xxiii., fasc. ii.-iii.)

1. Enrico Ballor, the "Hammerer." C. LOMBROSO.
2. The Compass Index. BELLOIN.
3. The Perception of Tactile Impressions. GRANDIS.
4. A Great Monomaniac—Girolamo Savonarola. PORTIGLIOTTI.
5. Voluntary Mydriasis and Epilepsy in a Man of Genius. LOMBROSO AND ANDENIRO.
6. The "Crime" of Striking. LASCHI.

1. *The Hammerer.*—This presents an analysis (psychological as well as anatomo-physiological) of a criminal who killed with a hammer two persons. It is the author's intent to prove that, even in the absence of marked physical stigmata of degeneration, the presented defects of the visual, motor, and above all, the moral functions stamp the subject as a born criminal. Hereditarily was found in the father a shiftless drunkard. one of the brothers a veritable subject of moral insanity, the mother dolicocephalous, with stenocrotaphy and prognathism, a spastic paralysis of the muscles of the cheeks and tongue, migraine and vertigo. It is also to be noted that the criminal had evidently suffered from an attack of meningitis and polio-encephalitis in his tenth year (before which time, by the way, he was a good, normal child). This illness seems to have left ineradicable and most important consequences, such as abolition of reflexes, impaired pupillary reaction, anomalies of the visual field, peripheral scotoma, and a tendency to excessive physical development as well as decided criminal propensities.

2. *The Compass Index.*—A description of an anthropometric instrument.

3. *The Perception of Tactile Impressions.*—A series of preliminary experiments in which pressure on the cutis was substituted by the induced electric current. Special attention is drawn to the relation between tactile sensibility to psychical function.

4. *Girolamo Savonarola.*—Will be abstracted when completed.

5. *Voluntary Mydriasis.*—This is an interesting account of a subject whose anamnesis, present state and anthropometric measurements, fully bear out the authors' contention as to his abnormality. The most singular feature of the case is the individual's faculty of voluntarily dilating his pupils, increasing the vascular pressure, and accelerating the cardiac movements; he could double the pressure while the pupillary dilatation was invariably accompanied by acceleration of the cardiac pulsations. The intervals between severe epileptic fits are characterized by extraordinary mental acuity, especially as to sociological studies. He also possesses a truly remarkable memory, especially for ideas, and a great facility for acquiring foreign languages; suffers to some extent from folie de doûte. With the above mentioned dilatation of the pupils there takes place a rapid contraction of the zygomatic muscles, and the patient grows pale and trembles; at the same time there appears strabismus. The authors assume the possibility of a vasomotor phenomena only to a certain extent voluntary, for a similar condition is observed in some artists, as for instance, the great Italian tradegienne Duse, who blushes and grows pale at will.

6. *The "Crime" of Striking.*—A critical survey of the history of labor organizations in several European States, the various legislative measures affecting them, especially as regards the workmen's rights to strike; of considerable interest to the student of sociological problems.

ALEX. ROVINSKY.

The Journal of Mental Science.
(1902. Vol. 48, No. 202, July.)

1. Report of Tuberculosis Committee of the Medico-Psychological Association of Great Britain and Ireland.
2. Toxemia in the Etiology of Mental Disease. Discussion by T. S. Clouston and others.
3. Surgical Treatment of Delusional Insanity based upon its Physiological Study. T. Claye Shaw.
4. Sleep in Relation to Narcotics in the Treatment of Mental Disease. Henry Rayner.
5. Notes in Some Cases of Morphinomania. Robert Jones.
6. Evolution of Delusions in Some Cases of Melancholia. Lionel Wetherly.
7. Pupillary Symptoms in the Insane and their Import. T. P. Cowen.
8. Prophylaxis and Treatment of Asylum Dysentery. N. H. Macmillan.
9. The Psychiatric Wards in the Copenhagen Hospital. Knud. Pontoppidon.
10. Clinical Notes and Cases.

1. *Tuberculosis Among the Insane.*—This subject has been widely discussed by the English alienists and the Report of the Medico-Psychological Association is given here in full. The work is mainly that of Dr. Eric France, who was appointed honorary secretary to the committee. The dangers are very fully pointed out and a most careful and comprehensive series of statistical tables emphasizes the great importance of the inquiry. Space permits only a short summary of this report. The conclusions given are: that phthisis is prevalent in the public asylums to an extent which calls for urgent measures. A very large number of cases of phthisis have acquired that disease after admission to the asylum. The special causes for this increased prevalence of phthisis in the asylums are: Overcrowding, with consequent insufficient day, and especially night, cubic space per patient; insufficiency of hours in the open air; defects in ventilation and heating; uncleanly habits and faults in dietary.

The *means of prevention* suggested are: Early diagnosis, isolation of all phthisical cases, limiting the size of future asylums, checking overcrowding, increasing day and night cubic space, restricting number of beds in dormitories, increased and more thorough natural ventilation per patient, stopping of promiscuous spitting, supervision of dietary, properly constructed hospitals and sanitoria, or these failing, temporary isolation hospitals or special wards and airing courts set aside for the purpose.

2. *Toxemia in the Etiology of Mental Disease.*—This is an interesting discussion of the etiological doctrine of toxemia in mental disease. Pathological workers are considered to hold more to the auto-intoxication theory than clinical observers. Dr. Clouston in opening the discussion suggests a number of queries which must be satisfactorily answered if the causative factors are to be found. The influence of mental factors is one of the first: consciousness and mental states are modified in sleep, the hypnotic state, etc., why may not exaggerations in these physiological processes be concerned in the insanities? The abrupt onset and equally sudden termination of many insanities seem inconsistent with known toxic activities: the specific reactivity of cortical cells to purely sensory stimuli exhibits very wide variations. Morbid activity or abnormal torpor might bring about such insane reactions and the

theory of toxemia is unnecessary. Ancestral hereditary reactivity is pointed out as one of the most potent factors in determining abnormal mental life, especially when faulty heredity renders a nervous system peculiarly susceptible, even to the slightest stimuli; developmental and involutional insanities, such as are seen at puberty and in senility surely do not need a toxic hypothesis; many brains, by a natural development of their original characters, determine a later mental disease; immunity correlatives should be borne in mind; elevations in temperature are not necessarily the reaction to a bacterial or toxic agent. Toxemia might be an element in many insanities, 'tis true, but is it not more scientific to regard it as a secondary activity, rather than a primary one? The medical psychologist holds that the cerebral cortex is the real controlling center of secretory and excretory processes and of nutrition, and in its action or inaction might cause or counteract those states of non-resistiveness through which injurious bacteria and toxins were developed or destroyed. In syphilis, alcoholism, rheumatism and other similar states the toxic element is recognized, in other insanities the causation is best arrived at from other points of view: in short that insanity is a protean disorder and no single etiological factor is sufficient to explain these.

Dr. Ford Robertson spoke of the toxic theory but added no new features to the discussion. His definition of toxemia is interesting although from a pharmacological side very indefinite and unsatisfactory: "Any chemical substance which on being brought to a cell and taken up by it, caused disorder of its metabolism, was a toxine for that cell." Melancholia he regards as essentially a toxemia. He develops a few straw bail arguments, assuming, for instance, that neurologists regarded tabes as a disease of the cord, or that insanity was a disease of the brain, etc. No thinking alienist or neurologist ever reasons thus, nor have they for many a year.

Dr. G. R. Wilson gives some sound remarks on the breadth of the subject practically bearing out Dr. Clouston's closing remarks. Dr. Yellowlees emphasized the mental influences with which every alienist is familiar but the pathologist would seem to disregard.

Dr. Bruce raised the question, "Do the mental symptoms precede the physical or the reverse?" a question which the modern physiological psycho-pathologists would, we believe, find unnecessary and non vital.

Dr. Marr writes that when actual experimental proof of the relationship can be shown then there will be time to consider the theory..

3. *Surgical Treatment of Delusional Insanity.*—Dr. T. C. Shaw, adopts the thesis that disease of the brain is never universal, but is apt to be localized and suggests an ideal for operative treatment of some of the delusional insanities on the lines of operative interference in Jacksonian epilepsy. By a careful study of the symptoms, it is important to try to find a primary lesion focus, if possible. The problem of localization was one in need of thorough investigation. He had nothing definite to offer but the paper is suggestive and timely.

4. *Sleep, Narcotics and Mental Disease.*—Dr. Henry Rayner limits his consideration of sleep to such aspects of it as are related to the question of the treatment of sleeplessness by narcotics; to consider whether the state of narcotic sleep or narcotic stupor is as reparative as normal sleep, and whether the relief of the symptom sleeplessnss by the use of narcotics may not be too dearly purchased by the harm done in other directions. He concludes from a theoretical consideration of the pharmacological action of the various hypnotics, etc., that more harm is apt to result from the use of these agents than good. His arguments really resolve themselves into a question of his own clinical

experience and this has led him gradually to discard the use of narcotics, altogether in narcotic dosage, and only at rare intervals in the hypnotic form, and then only in the form of bromide, or a small quantity of alcohol.

5. *Morphinomania.*—Dr. Robert Jones, of Claybury Asylum, London, gives a short account of eight cases of insanity resulting from the habitual use of opium or morphine. Of the forms of insanity noted, the depressed insane states were commonest. Three males were melancholy and one maniacal; two females were melancholy, one was suffering from delusional insanity and one from mania. In the matter of treatment he advocates sudden withdrawal.

6. *Evolution of Delusions and Melancholia.*—Dr. L. Weatherly says that in many melancholiacs the delusions have not had their origin in the imagination only, but they have been evolved from some definite reality. A tendency to brood over these realities becomes more and more rooted and they take full possession of the mind, overmaster the will, pervert the reason and gradually develop into a systematized delusion. To treat these cases half measures are of little value and the author advocates the following: (1) Cut the patient adrift from his associations and place in a new environment; (2) build up the physical health; (3) occupy the mind by diversified walks and amusement, neither fatiguing, nor permitting time to hang heavy; (4) gain the patient's confidence. Never deceive him; (5) never allow the attendants or nurses to attempt to get their patients to do what they wish by false promises, or by agreement with them in their insane ideas.

7. *Pupillary Symptoms in the Insane.*—Dr. T. P. Cowen practically limits his paper to the pupillary symptoms as seen in general paresis. Two classes of pupillary abnormalities are observed in insanities, he writes: (a) inconstant variations; (b) persistent or wholly progressive impairment. The former are alone met with in cases other than general paresis and senile dementia, whereas both classes of symptoms are present in the latter organic disease. In general paralysis the first eye symptom is a slight inequality of the pupils with a sluggishness on the part of the larger pupil to contract to a subdued light, accompanied by an absence of the usual dilatation following cutaneous stimulation, while all the other reactions are perfectly normal. Of the symptoms in detail he says: *Size*—The pupil is more often dilated, which dilatation may be unilateral or in both. *Inequality*—This is an early symptom and not necessarily associated with impairment of the reflexes. *Contour*—One or both may be irregular. They may be oval above, below, or in both situations. *Mobility*—He finds higher figures than those of Bevan Lewis, 13.6 per cent. *Light Reflex*—In 33 per cent. of his cases both pupils were fixed; in 38 per cent. one or other was fixed or sluggish; 23 per cent. showed sluggish reaction in both; 3 per cent. were normal; 2 per cent. showed a constant oscillation.

These affections of the pupil are not confined to true paresis but may be seen in alcoholic, syphilitic and saturnine pseudo-paresis. In the remissions of paresis the pupillary symptoms may disappear. This recovery, he notes, he has never observed with the markedly myotic pupil.

8. *Asylum Dysentery.*—Dr. Macmillan gives his experiences with this disease at Claybury. The means adopted to prevent its spread are isolation, and asepsis in regard to all the details of the patients, food, dress, etc. Especial attention should be devoted to the relapsing cases. So far as active treatment is concerned, no specific has as yet been found. In uncomplicated cases the treatment is expectant. In the more severe cases castor oil dr. ss-iss, with or without opium, or magnesium sulphate, are exhibited. The newer proteid foods, as tropan,

plasmon, etc., are of service. Alcoholic stimulation may be necessary. In the excessive diarrheas, after the disappearance of blood, subnitrate of bismuth and opium are valuable. Thirst is best relieved by cracked ice; pain is relieved by turpentine stupes, and tenesmus is remedied by small enemata of opium and starch.

9. *Psychiatric Wards at Copenhagen.*—A short account of the buildings used in the Copenhagen general hospital for psychiatric purposes. JELLIFFE.

Jahrbücher f. Psychiatrie und Neurologie.

(Vol. 21, Nos. 1 and 2, 1902.)

1. Changes in the Motor Cells of the Spinal Cord After Resection and Tearing out of the Peripheral Nerves. E. STRAUSSLER.
2. Migraine Psychosis. KRAFFT-EBING.
3. Clinical Discussion of So-Called Traumatic Syringomyelia. R. KNIEBOCK.
4. The Clinical and Anatomical Facts in a Case of Cerebellar Tumor. M. PROBST and K. von WIEG.
5. A Case of Isolated Softening of the Gyrus Hippocampus and Its Immediate Vicinity—Secondary Denegeration. E. BISCHOFF.

1. *Changes in Motor Cells of Spinal Cord.*—Although for a decade the ganglion cells have been most earnestly studied, the results cannot be said to be satisfactory. The difference of opinion has extended even to the fundamental principles which Nissl promulgated, namely, that changes in the nerve cell take place when there is a break in continuity between the nerve and the muscle cell, irrespective of how the break occurs; that is, whether the nerve is simply cut, resected, or torn out with violence. Van Gehuchten and others found that simple interruption of continuity caused no cellular changes, while De Buck and Vanderlinden were not able to demonstrate cell changes in a number of experimental amputations on the extremities of animals. Marinesco has repeatedly called attention to the fact that a difference exists accordingly as the nerve is torn, or is simply cut. With this question came also another, that of retrograde degeneration, which, first announced by Nissl, was seized upon eagerly by other investigators as an explanation of the various different findings in experiments of this nature. The author of this paper planned a series of experiments which had for their main purpose the determination of the difference in the cells of the cord which is found when a simple break in continuity of a nerve fiber exists and in one in which the break is caused by the forcible tearing out of the nerve. Cats were used for the experiment and the brachial plexus or the sciatic nerve was selected as the object of the experiment. The results after resection were the following: From five to thirty days after the operation a gradual progressive alteration of the ganglion cells was observed. This alteration was especially marked in the tigroid substance. It was accompanied by swelling of the cells, with little or no alteration of the nucleus. The cell branches were broken. After tearing out the spinal nerves, cellular changes appeared in twenty-four hours, which at first were limited to the tigroid substance. Within five days definite changes in the nucleus were added, which in many cases resulted in the destruction of the same. Thirty days after the operation the process is concluded with the destruction of more than half the cells. Tearing out of the spinal nerves causes a lesion of the anterior column, which leads to a reactive inflammation, with proliferation of the glia cells. The author ends this study with the following general conclusions: (1) The changes which follow an uncomplicated simple resection of the spinal nerves

are not directly of importance to the life of the cell and to the trophic activity of the same. (2) Tearing out of a nerve is followed by severe degenerative changes in the cell, which is not dependent upon the break in continuity nor on the location of the lesion, but upon the reactive inflammation caused by the trauma. (3) The cell changes which follow simple break in continuity cannot be explained by the fact of the degeneration in the proximal portion of the nerve fiber following the lesion. Severe cells changes depend upon other causes than simple break in conduction. To offer as an explanation for the cell changes retrograde degeneration in the Nissl sense is of no value.

2. *Migraine Psychosis.*—The fact that a migraine attack is sometimes accompanied by a transitory psychosis led Krafft-Ebing to think that perhaps the relation is not a matter of chance, but that the psychosis was a direct result of the migraine. The psychical disturbances accompanying an ordinary migraine are too slight to be of great importance. They are simple anxiety, depression, fear, etc. Those migrainous attacks which develop upon an epileptic basis are complicated with certain forms of psychosis, which may be considered either an epileptic equivalent or a manifestation of post-epileptic insanity. Especially to be mentioned is the so-called ophthalmic migraine, which more frequently than the other forms accompanies an epileptic attack. Towards the elucidation of this question, Krafft-Ebing reports nine cases, careful clinical histories of which are included in this article. From these observations and others, Krafft-Ebing believes that there is no proof at present that such a form as an independent hemicranial psychosis exists. The question must be regarded as still open. The variety of such psychoses is a proof of their etiological complexity. It is only in cases of ophthalmic migraine, or that complicated with Jacksonian epilepsy, that transitory psychoses have been observed.

3. *Traumatic Syringomyelia.*—This paper by Kniebock is a valuable and painstaking review of all the published cases of so-called traumatic syringomyelia, for the purpose of testing the accuracy of the term and for the purpose of finding out the place which trauma occupies in the causation of this disease. It was Leyden, in 1874, who first called attention to a cystic degeneration of the cord following trauma. Since then numerous cases have been reported in literature. It is with a critical estimation of these cases that this paper has to do. The author defines syringomyelia as a condition in which chronic progressive foci of disease appear in the spinal cord. Such foci are situated in its center with a longitudinal direction, and the process is accompanied by glia hypertrophy, severe vascular changes, and by the formation of cavities. This definition is to be understood in its narrower sense only, namely, that of syringomyelia gliosa, as it is only in this form that a clinical diagnosis *intra vitam* can be made. Other affections which have to do with cavity formation, such as hydromyelia, sclerotic scars following injuries, do not come within the limits of this definition. The term traumatic syringo-myelia should be given only to those cases in which the disease is caused by trauma, and in which the symptoms, both clinically and anatomically, are characteristic of this disease. This term should neither be given to those cases which are really not syringomyelia (gliosis), not to such in which the trauma is merely a secondary factor. One hundred and forty cases are collected from the literature; nine cases of the author's own are likewise considered. Some of the data derived from a study of this large material are as follows: The first division consists of cases which were diagnosed as traumatic syringomyelia and none of which satisfied the conditions of the above definition. This series illustrates how a centrally-located traumatic lesion can produce a symptom

complex very similar to that of syringomyelia but differing from it essentially in the lack of a chronic progressive course. A second group consists of six cases in which the symptoms were produced by a cystic degeneration of scar tissue following traumatic destructive foci. Here the cavities are mere softening cavities, produced in part by beginning absorption of the softened substance in the center of the focus and in part artificially by post-mortem manipulation. A further group is noted of severe traumatic spinal lesions in which changes in the spinal canal followed. A second division consisted of clinical observations in syringomyelia in which the traumatic nature can be set aside with great certainty. In this division it is not the diagnosis of syringomyelia which is at fault, but the term traumatic, as significant of an etiologic factor. To this group belong also the cases which result from peripheral trauma. There are two groups in this division, one consisting of cases in which too long an interval has elapsed between the trauma and the beginning of the symptoms, and the other where the interval was entirely too short, in one case less than one week. All these cases are carefully described and a critical study of them is added in each instance. The author concludes as the result of the study of this material that after careful examination of all the known cases, no certain example of traumatic syringomyelia, that is pure (*per se*), caused by a powerful trauma, can be found. Contrary to the opinion of many authors, Kniebock holds that the existence of a simple chronic and hematomyelogenous (that is the condition that follows a paralysis, especially paraplegia, caused by apoplexy) traumatic syringomyelia is a matter of much doubt. Outside of the clinical interest in this subject there is also a forensic side. It has always been thought a matter of certainty that a traumatic syringomyelia existed; now that this is not very likely, the question of damages in a case of syringomyelia developing after trauma must take into consideration either the likelihood of a latent syringomyelia or some special predisposition on the part of the patient. A very complete and well-arranged bibliography is found in this paper. The article is in every sense an admirable one and adds a great deal to our knowledge of this subject.

4. *Clinical and Anatomical Facts in a Case of Cerebellar Tumor.*—It has come to be believed that the function of the cerebellum has not only to do with regulation of voluntary movement of the body, but also serves as a sensory-motor organ. Clinical observation in increasing number has strengthened the latter hypothesis. Experimental evidence as to the function of the cerebellum cannot be directly applied to human cases; therefore each case of cerebellar tumor should be more carefully studied clinically and pathologically. To accomplish the latter purpose it is necessary to make a very exact serial section of the whole cerebellum. The case upon which the article is based is that of a boy, 12 years old, with no hereditary history, who had suffered for some months from vertigo, headache and vomiting. On examination the left side of the face was less responsive to stimulation than the right; the pupils were wide and did not react to light and accommodation. There was a papillitis with beginning atrophy. In the arms and legs there was slight ataxia. Romberg symptom, knee-jerks increased on both sides. The patient was passive and indifferent.

The anatomical substratum for this symptom-complex was a glioma of the cerebellum, which had grown to the middle lobe and the left lateral lobe, and had caused a hydrocephalus, with symptoms of increased intracranial pressure. A careful description of the microscopical findings in the serial sections is given in the article, which is illustrated by microphotographs.

5. *A Case of Isolated Softening of the Gyrus Hippocampus and Its Immediate Vicinity—Secondary Degeneration.*—The anatomical connection of the hypocampal gyrus which in all probability has to do with the cortical smell center, has been very little studied. There is no report in literature of the study of an isolated lesion of this region. A short clinical résumé of the case is as follows: Woman, aged 65 years. For twenty-three years she has had epileptic attacks from three to six times each month. Some of these attacks were followed by epileptic stupor or psychosis. She died of marasmus. During life careful examination revealed nothing abnormal in respect to the special sense nerves or to motility or to sensibility. Autopsy showed a moderately diffuse cortical atrophy and a focal disease on the inferior and internal surface of the right temporal lobe. Serial sections of the frontal and temporal lobes were made; the focus was found to be cyst. This case seems to offer definite proof of the fact, which has been assumed already, in studies of comparative anatomy and embryology, that the nerve fibers of the fimbria arise exclusively in the gyrus hypocampus, the uncus and the Ammons horn, that the fornix is made up of two portions in its dorsal knee, the fimbria portion and the so-called fornix longus, the latter arising from the corpus callosum. It appears that in man the calumna is an uncrossed communication between the Ammon's horn and the corpus mammillaria of one side. The fibers of the fornix longus end, as this case shows, in the neighborhood of the septum pellucidum, the fimbria fibers in part uncrossed in the lateral nucleus of the corpus mammillaria and in part crossed in the region of the septum pellucidum.

SIDNEY I. SCHWAB (St. Louis).

Nouvelle Iconographie de la Salpêtrière.

(No. 2, March-April, 1902.)

1. Affections of the Cauda Equina and the Lower Segment of the Cord. F. RAYMOND.
2. The Anatomy of the Lacunæ of Disintegration of the Brain. JEAN FERRAND.
3. Degenerative Infantilismus, Complicated with Dysthroidea of Puberty (Brissaud's Type). E. DUPRÉ and PHILLIPE PAGNIEZ.
4. Congenital Absence of the Greater and Lesser Pectoral. A. SOUQUE.
5. Little's Syndrome. GASTON DANIEL.
6. The Sense of Attitude. BONNIER.
7. Note on the Radicular Topography of Myelopathic Muscular Atrophy. CESTAN and HUET.

1. *Affections of the Cauda Equina and of the Lower Segment of the Cord.*—The anatomical limits of the conus terminalis have been fixed arbitrarily by anatomists. Recently Müller of Erlangen has found a basis that seems to be strictly scientific. That portion of the cord which gives rise to the last three pairs of sacral nerves and to the coccygeal nerve differs essentially in its histologic constitution from the adjacent structure. Raymond founds his limit upon clinical grounds in the following way: It comprises all paralytic symptoms of the bladder and rectum, associated with impotence in the male and an anesthesia limited to the anus, perineum, scrotum, penis in men, to the vulva, labia majora, and to the urethro-vesical mucous membrane in women. To this group of symptoms another may be added as it is frequently met with, consisting of a motor paralysis of the posterior region of the thighs, the legs, and the feet; (2) an anesthesia, total or dissociated, which occupies the median part of the posterior aspect of the thighs and legs, the external edge of the leg and foot, sometimes all the surface of the foot with the exception

of a narrow band adjacent to the internal border; (3) abolition of the Achilles reflex. In other words, it means a sensory-motor paralysis of the sacral and sacro-coccygeal plexus. The following case is given as an illustration: Man, 54 years old. As the result of a fall from a moving train the following symptoms appeared seventeen days after accident: (a) incomplete motor paralysis of the inferior extremities; (b) vesico-rectal paralysis; (c) functional genital symptoms; (d) trophic disturbances; (e) sensory zone of anesthesia—ano-perineo-rectal, which extended upon the neighboring buttock region and upon the posterior portion of the thigh. Zone of hypo-asthesia limited to the external border of each foot on the dorsum. Subjective symptoms of pain, caused by passive motion of the lower extremity. The treatment of these cases may be divided into main divisions: symptomatic and causal. The latter is by far the more important, and under this head may be considered the three following possibilities: (a) traumatism, that is, a luxation of a vertebra or a fracture, causing compression of the spinal roots; (b) in the absence of an antecedant traumatism, the possibility of a specific infection must be considered; if present, it is probably an extradural process, a pachymeningitis specifica, which reacts very favorably to antiluetic treatment; (c) in cases where no trauma or specific infection is found and yet where the symptoms are the result of a circumscribed lesion, acting upon the cauda equina by means of compression, the symptoms of pain being especially marked, then the possibility of a tumor is very great. In such cases the surgical intervention, looking to the extirpation of the tumor, is not only indicated but is necessary.

2. *The Anatomy of the Lacunae of Disintegration of the Brain.*—The lacunae of cerebral disintegration were little studied before Pierre Marie took them up. This paper is based upon material collected in the laboratory of Bicêtre. The study of the anatomy underlying the loss of cerebral substance, the most frequent cause of the hemiplegia of old people, is the chief purpose of this paper. The chief results of this investigation may be summarized under the following heads: Macroscopic anatomy of the lacunae. The most frequent seat of the lesion is in the gray nuclei of the brain. Of one hundred lacunar lesions, eighty-seven were found in the central gray nucleus, and only thirteen in the white substance. Under histology of the lacunae, the principal facts noted are—whatever the size or the location of the lacunae may be, the process of their formation consists of two steps, first the miliary lacuna, which may be considered as the preparatory step. It is characterized by the fact that around a cerebral arteriole the tissue becomes thin but adherent to the vessel. In the second stage there is a formation of a cavity between the central vessel and the cerebral parenchyma. Lesions of the vessels. The central vessel is usually one of moderate calibre; sometimes it is an arteriole, but its walls are always very much thinned. Pathological change is found chiefly in the muscular layer. Connective tissue elements replace muscular fibers. This substitution takes place very gradually without any leucocytic infiltration and without any phenomenon indicating that the vascular walls have been the seat of any inflammatory reaction whatever. This change is not characteristic of cerebral vessels but is common to all senile arteries. The walls of the lacunar cavity are formed by the cerebral tissue itself, very little altered. Contents of the lacunæ. There are two sorts of elements, one from the cerebral parenchyma itself, and the other from the blood vessels. Sometimes the process of disintegration can be arrested by the cicatrization of the cerebral tissues and the proliferation of neuroglia can fill the cavity so that no trace of beginning cavity formation can be demonstrated.

3. *Degenerative Infantilismus.*—An account illustrated with photo-

graphs and radiographs of a case of degenerative infantilism. The interest in this observation consists in the successive factors which caused the developmental arrest of this child. Among them are to be noted alcoholic heredity, premature birth, retardation of dentition, the late appearance of speech and intelligence. This group tends to show a state of hereditary infantilism of toxic origin. An attack of typhoid fever at five years of age furnished the infectious element acting to favor the retardation of growth. At the age of fourteen there developed a myxedematous condition together with appearance of a rudimentary menstruation.

4. *Congenital Absence of the Greater and Lesser Pectoral.*—The congenital absence of these muscles is rarely observed. Such a case is described in this article, which contains a beautiful photograph of the case, with a radiograph of the right hand, which shows a curious lack of development in regard to size.

5. *Little's Syndrome.*—The author calls attention to the confusion which at present exists in the group of symptoms known under the name of Little's disease. In 1852 Little published a series of observations relating to the deformity of the human frame. These symptoms had been known before then under the term spasmodic tabes. With the advance in histological study these various symptoms became known as Little's disease. Van Gehuchten has given the characteristics of Little's disease as, first, premature birth; second, spasmodic contractures with absence of cerebral lesions; third, tendency to spontaneous and progressive cure. The whole affection is due to the lack of development of the pyramidal tract. The author gives a critical résumé of these characteristics. In regard to the first, Little's disease has been observed in infants born at term, and has been even known to appear two years after birth. In regard to the second, it is noted that these symptoms are not very well defined and are just as well applicable to the paralyses and paraplegias of infants. It seems almost impossible to imagine a paraplegic contracture or dystrophy without intelligence being indirectly affected. In regard to three, this is not constant at all; while some cases show a distinct tendency towards improvement, others show just the reverse. As to four, the pyramidal tract at best is not completely formed until the ninth or tenth month, as is shown by histological study and by the persistance of the Babinski sign. Nevertheless the average baby does not present in the first months of its life any evidence of contracture or incoördination of the extremities. It is probable that under this term at present a diversity of conditions may be understood and that no definite anatomical or clinical meaning can be attached to it.

6. *The Sense of Attitude.*—A study in the psychology of the sense of attitude, which Bonnier defines as that quality which informs us concerning the relative position of every part of ourself to ourself. Impossible to abstract. SIDNEY I. SCHWAB.

Rivista di Patologia Nervosa e Mentale.

(June, 1902, v. vii., fasc. 6.)

1. An Anomaly of the Cerebellum. DON. D. ROVEW AND B. DE VECCHI.
2. Complete Ablation of the Thyroid Body in a Dog. G. CORONEDI AND G. MASCHETTI.
3. A Contribution to the Pathogenesis of Exophthalmic Goiter. ETTERE TEDESCHI.

3. *Exophthalmic Goiter.*—The following conclusions are arrived at by the author after a series of experiments on rabbits and dogs:

1. Lesions of the restiform bodies (especially in their anterior portions) produces symptom complex of morbus Basedowi.

2. In animals thus injured the symptoms of the disease are liable to recur again and again in part, or in their entirety, even after they have once disappeared.

3. In animals almost entirely deprived of the thyroid body, lesion of the restiform fails to produce the symptom-complex of Basedow's disease.

4. In animals in whom a lesion of the restiforms resulted in the production of the disease, the excision of the thyroid body causes either a diminution or a disappearance of the greater part or even of all the symptoms of this disease. ALEX. ROVINSKY.

Neurologisches Centralblatt.

(Vol. 21, 1902, No. 11, June 1.)

1. On Central Eye-Muscle Tracts. J. PILTZ.
2. Spinal or Muscle Atrophies, Which? TOBY COHN.
3. Further Concerning Asthenic Paralysis. E. FLATAU, S. GOLDFLAM.

1. *Central Eye-Muscle Tracts.*—Piltz has studied by means of the Marchi method the secondary degeneration resulting from removal of the cortical areas inervating the eye-muscles of the dog. After removal of the area in the frontal lobe he found degeneration in the neighborhood of the lesion, corpus callosum, anterior limb of the internal capsule, lenticular nucleus, stratum intermedium of the tegmentum, and the internal medial division of the pes. After extirpation of the Munks region F in the parietal lobe secondary degeneration was found in the cingulum, the longitudinal subependymal fibers near the roof of the lateral ventricles, corpus callosum, internal capsule of the same side, optic thalamus, subthalamic region and lateral division of the pes.

2. *Spinal or Muscle Atrophies?*—To be continued.

3. *Asthenic Paralysis.*—Goldflam reports in extenso eight cases of asthenic paralysis and discusses the etiology, pathology, pathogenesis and treatment. In the only case in which an autopsy was obtained, the pathological examination revealed a perfectly normal condition of the central and peripheral nervous system. A lymphosarcoma was found in the anterior mediastinum with metastases into the muscular system. Notwithstanding the involvement of the muscular system in this case and that reported by Weigert, he is inclined to regard the causative factor as toxic, and the symptoms as a result of intoxication of the central nervous system. Thymus therapy in his cases was not at all successful or satisfactory.

(Vol. 21, 1902, No. 12, June 16.)

1. New Contribution to the Pathological Anatomy of Amyotrophic Lateral Sclerosis. A. VON SARBO.
2. On the Direct Antero-lateral Pyramidal Tract. W. G. SPILLER.
3. Fecal Vomiting in Status Epilepticus. H. GOLTZ.
4. Spinal or Musclar Atrophy, Which? TOBY COHN.

1. *Amyotrophic Lateral Sclerosis.*—Von Sarbo found in a case of amyotrophic lateral sclerosis, studied by the Marchi method, marked degeneration of Clarke's columns and also of the direct cerebellar tract; the degeneration of the latter he was able to follow through the restiform body into the superior vermis of the cerebellum. There was also degeneration of the posterior longitudinal fasciculus, the hypoglossal and facial nuclei and the usual lesions found in the spinal cord. He was not able to find the degeneration in the cortex cerebri, as did Spiller in his case.

2. *Ventro-lateral Pyramidal Bundle.*—Spiller maintains the identity of the tract discovered by him and known as the ventro-lateral pyramidal bundle, as distinct from the Helweg's bundle. This position is confirmed by the relation of these bundles of fibers in the spinal cord and has been confirmed by the work of Barnes.

3. *Fecal Vomiting in Status.*—Report of a case of epilepsy in a woman of fifty-one years who during epileptic status had repeated attacks of fecal vomiting. This was considered to be due to a contraction of the musculature of the bowel during the epileptic attack.

4. *Spinal or Muscular Atrophy?*—Report of a border line case of muscular dystrophy or neural atrophy. The case followed the peroneal type, with muscular twitchings and evidence of degenerative changes. The author was unable to arrive at a positive diagnosis between the two conditions.

(Vol. 21, 1902, No. 13, July 1.)

1. Pathology of Tetany. Prof. A. Pick.
2. Silver Impregnation of Axis Cylinders. Max Bielschowsky.
3. Hysterical Blindness. H. Kron.

1. *Pathology of Tetany.*—A preliminary notice of a paper to be published in full in the *American Journal of Insanity* concerning the presence of calcification of the smaller cerebral vessels as a causative factor in the production of tetany.

2. *Silver Impregnation of Axis Cylinders.*—A new method for axis cylinders and nerve cell staining. The paper is not suited for abstracting on account of the complexity of the method.

3. *Hysterical Blindness.*—To be continued.

(Vol. 21, 1902, No. 14, July 16.)

1. Subcortical Origin of Isolated Muscular Movements. A Contribution to the Study of Tumors of the Corpora Quadrigemina and Observations on the Central Tegmental Tract. J. Sorgo.
2. A New Method of Preparing Histological Preparations. F. Reich.
3. Hysterical Blindness. H. Kron.

1. *Isolated Muscular Cramps.*—To be continued.

2. *Histological Technic.*—Macerated portions of fresh material, or sections of the same are shaken in a test tube and after separating the vessels, the pia, etc., the milky residue is centrifuged and treated by different staining methods.

3. *Hysterical Blindness.*—A report of two cases of hysterical blindness, with a résumé and classification of cases found in the literature.

D. J. McCarthy (Philadelphia).

Beiträge zur psychiatrischen Klinik.

(1902. Vol. i, No. 2, June.)

1. Analysis of the Ideational Content in Epileptic Dementia. Manfred Fuhrmann.
2. The Question of the Relation of Bodily and Mental Diseases to Each Other. Koester.
3. Contribution to the Knowledge of the Effect of Alcohol on the Motor Functions in Man. Hornung.

1. *Ideatation in Epileptic Dementia.*—The researches to determine the ideational content were conducted with three epileptics, the same experiments first being checked with normal persons. The method consisted of reading lists of words and having the patient react by in-

forming the experimenter of the ideas the words read (*Reizwort*) called up. The same process was repeated with the same words not less than four weeks afterwards, and the differences noted, especially the new associations. The number of new associations based on one hundred words gave a percentage indicating the extent of association *(associationsweite)*. In the normal individual this percentage varies from 75-95 per cent. varying naturally because of the individual's education; while 60 per cent. and under must without doubt be pathological.

The first patient experimented with showed an extent of association of 62.4 per cent., the percentage being least with abstract words. There was found also a great tendency to the frequent repetition of the same words indicating, the author thinks, a high grade dementia or an inhibition phenomenon like that seen in catatonia. Occasionally the patient responded with great suddenness, before the *reizwort* could have a chance to pass the threshold of consciousness. This the author calls "unconscious reaction" and thinks it characteristic of epilepsy.

The second case showed an extent of association of only 33 per cent. If, however, the experiments are confined to concrete objects the percentage is 44 per cent. as compared with two idiots who respectively showed 40.58 and 46.38 per cent. The simple, trivial, grammatical construction of the sentences used in reacting is also significant of the dementia the *reizwort* being used as subject or object with some simple verb as "is", "has", "gets", etc. The difference between the epileptic and the idiot lies in the fact that the former has knowledge of superior (*übergeordnet*) ideas, and grasps their meaning, making use of them correctly but reacts to these superior ideas as expressed in the *reizwort* in a very simple *(untergeordnet)* manner. The idiot, however, shows nothing of this possibility.

In the third case the extent of association reached 83.7 per cent. but it is noteworthy that this was a case of organic epilepsy (cerebral tumor), the first attack not occurring until the patient was 29 years old and he being able to support himself as a musician until his thirty-fifth year. There was noted in this case a monotony in the reaction, the same word often being used in each of the four series of experiments made to react to the same idea and the same emotional tone being called up by the *reizwort* on each occasion. The simplicity of grammatical expression was also prominent.

In summing up the author regards the "unconscious reaction" as the most characteristic feature of epilepsy. Added to this is the monotony of reaction together with its great slowness, not infrequently amounting to one-half and three-quarters of a minute. Very natural also is the rare appearance of the so-called clang association.

2. *Relation of Bodily and Mental Disease.*—The author, after commenting on the fact that in general intercurrent physical diseases have been held to have a beneficial influence upon existing psychoses, arrives at the following conclusions from a study of several cases. (1) The prognosis of a bodily disease is often unfavorably influenced by a contemporaneous mental disturbance. (2) Commonly the psychosis undergoes a modification due to the influence of the bodily disease which makes its symptoms appear in an unfavorable light so that the bodily disease may appear to have a favorable influence upon the prognosis of the mental disease. This is well shown by the author's fourth case in which the symptoms were of a well marked paranoid type. (3) The prognosis of a mental disease complicated by a bodily disease is independent of the bodily disease as soon as we have to deal with an independent, established psychosis and not a simple symptomatic psychosis. This is illustrated in a patient who had a case of pneumonia compli-

cated by a beginning catatonia. The psychosis, the author thinks, might have been initiated (*ausgelöst*) by the pneumonia.

3. *Effect of Alcohol on the Motor Functions.*—This article was suggested and is in a way supplementary to the article in the first number of the *Beiträge* by Alber. In taking curves of the movements of the leg due to eliciting the patellar tendon reflex the author found that there was often a tendency for the leg to oscillate pendulum fashion before coming to rest. This tendency was increased by anything which distracted the mind (reading) and was also increased by alcohol. It does not occur to any extent in normal persons and the author concludes therefore that it is due to lack of inhibition. The article is illustrated by reproductions of the various kymographic tracings studied.

Wm. A. White (Binghamton, N. Y.).

MISCELLANY.

Neuralgia of the Bladder. G. Frank Lydston. (Jour. Amer. Med. Asso., Aug. 23, 1902).

Crystalgia has been classified as follows: (1) Crystalgia with lesions of the urinary apparatus, *i.e.*, urethra, bladder and kidneys. (2) Crystalgia with lesions of neighboring organs, such as the testicle, prostate, rectum and anus. (3) Crystalgia incidental to ataxia or general paralysis. (4) Crystalgia associated with diathetic conditions, as rheumatism, gout or malaria. (5) Essential crystalgia. Neurotical study is properly directed to the crystalgia associated with diseases of the cerebro-spinal axis. Vertical pains are common in the pre-ataxic period of locomotor ataxia. Crystalgia occurs in the hysterical or chloranemic patients. Neuralgia of the bladder is of variable intensity and radiates in different directions through the hypogastrium, groins, testes, thighs perineum, rectum or back. If definite lesion, such as calculus or other disease is absent, essential crystalgia is to be diagnosed corresponding to neuralgia of other organs. W. B. Noyes (New York).

Amnesia. S. D. Hopkins (N. Y. Med. Jour., Aug. 30, 1902).

Amnesia is frequently seen in the various insanities, organic diseases and concussion of the brain, in epilepsy, in somnambulism, in the hypnotic state and following fright. Amnesia may be partial or complete. In the former case memory is only lost to certain objects or groups of objects, without involving other portions of memory; in the latter the memory may be completely lost for both present and past events, as is seen in organic diseases of the brain and spinal cord. A normal mind must not only be able to call up past recollections and to register impressions to which the present attention is called, but also be capable of correlating the present with the past. Inability to do this will produce a form of amnesia known as double consciousness. A case was reported by the author of amnesia from July 4 to September 21, 1900. The patient's last recollection was in a clubhouse near New York. He awoke in Denver, in workingman's clothes bought in Philadelphia and Chicago. His hands bore marks of hard labor.

W. B. Noyes (New York).

The Education and Development of Neurotic Children. Graeme M. Hammond (The New York Medical Journal, Aug. 30, 1902).

The occurrence of infantile convulsions, chorea, night terrors and kindred disorders during infancy will determine a neuropathic disposition, apart from disease of the parents.

Toward the fifth or sixth year or upwards, the neurasthenic and hysterical temperaments begin to manifest themselves. Exceptional mental ability without corresponding physical strength is sometimes

the early and only manifestation of the neurotic temperament. Precocious and unusually clever children frequently fail to hold their own later on. With the advent of puberty, or even earlier, nervous disorders are most likely to show themselves. Sometimes fretful irritable temper is developed, and migraine or even epilepsy may appear. Masturbation or abnormal sexual conditions frequently begin at this time. The writer believes that the neurotic predisposition, like the tuberculous predisposition, may be entirely eradicated by a proper system of training. The diet of the neurasthenic child should be largely nitrogenous, but no overfeeding should be allowed. These children need to be taught self-restraint, control of the emotions and obedience with more firmness and insistance than is usually necessary with normal children. Mental application should be delayed until after the seventh or eighth year, and mental training generally not begun until the condition of physical health is firmly established. WM. B. NOYES (New York).

RESECTION OF THE CERVICAL SYMPATHETIC IN BASEDOW'S DISEASE. Belacescu (Archiv. f. klin. Chirurgie, Apr., 1902).

The author sets out to determine to what extent the cervical sympathetic plays a rôle in the production of Basedow's disease. Each of the principal and subsidiary symptoms may be shown to have some connection with an irritation of the cervical sympathetic. Exophthalmos, one of the most prominent symptoms, is believed by most authors to be dependent upon a stimulation of the cervical sympathetic, leading to an energetic contraction of the muscular cone, known as Müller's smooth muscle, at the posterior pole of the bulb. If this be true, section of the sympathetic would prevent the exophthalmos, which it does in all cases. The enlargement of the thyroid depends on an enormous dilatation of the vessels of the latter, which in turn is dependent on a stimulation of the vasodilator fibers of the neck and chest. The activity of the vasoconstrictor fibers is inhibited and the dilatation of the arteries causes hypertrophy of the gland. The theory of Jonnesco is that the struma of Basedow's disease is dependent on the increased activity of the thyroid elements and hypersecretion of the gland, which are dependent in turn on the permanent stimulation of the secretory fibers of the thyroid. Whichever of these theories is correct the resection of the cervical sympathetic causing section of the vasodilator, vasoconstrictor and secretory fibers, results in atrophy of the goiter. This in fact occurs in every case. Tachycardia is likewise to be attributed to irritation of the cervical sympathetic. The accessory symptoms seem to be dependent on changes in the circulation of the brain brought about by stimulation of the cervical sympathetic. These symptoms, consisting of tremor, sensations of heat, sweats, gastro-intestinal disturbances, and the nervous excitability so frequently seen in this disease, are, according to Jonnesco, dependent on a permanent cerebral anemia produced by a continuous stimulation of the vasoconstrictor fibers of the cervical sympathetic going to the brain. Extirpation of these fibers leads to cerebral congestion. The various operations on the cervical sympathetic for the relief of exophthalmic goiter are the following: (1) Simple division of the cervical sympathetic; (2) ablation of the cervical sympathetic by means of Jaboulay's operation, which, without a large incision, is devised to stretch and twist the nerve by means of artery forceps attached to its upper and lower ends; (3) simple stretching of the cervical sympathetic; (4) partial resection of the latter; (5) partial and extensive resection; and (6) total resection. Considering the results of the large number of operative procedures for the cure of primary Basedow's disease, the author concludes that total and bilateral resection of the cervical

sympathetic, which is known as Jonnesco's operation, is the most effective. The mere stretching and even the simple division of the nerve do not destroy all the connections of the sympathetic with the thyroid. The partial and extensive resection is applicable in only those cases in which the tachycardia is not pronounced. Although the direct surgical treatment of simple goiter by means of the almost total resection of the thyroid gland is not in itself a dangerous operation, in the struma of Basedow's disease it is very serious and is accompanied by many disadvantages.

JELLIFFE.

A SCHEME FOR THE ANTHROPOLOGICAL EXAMINATION OF INSANE PATIENTS WITH THE RESULTS OF ITS APPLICATION TO CONTROL PATIENTS. E. Goodall (Br. Med. Jour., Oct. 26, 1901).

The author says that the best work has been done in the United States and Italy. The full application of his scheme to ruly patients occupies from three to four hours. He calls special attention to the fact that there can be no intelligible or useful statistics created without a normal standard to judge them by, and this must be gathered from measurements made on normals born and bred in the locality from which the insane come. The main objects of comparing the normal and abnormal standards are as follows: (1) To ascertain what conditions constitute deviations from the normal; this is not yet known. (2) Which of these are most common in the insane. (3) Whether they are grouped about the head and face. (4) Whether there exist stigmata by which the gravity of a case can be measured. (5) Whether there is a correlation of any value between certain stigmata and definite mental disorders. (6) The relation of inheritance to mental disorders. (7) Relation of stigmata to prognosis.

JELLIFFE.

BASEDOW'S DISEASE. C. L. DANA (N. Y. Med. Jour., June 14, 1902).

This author believes that the primary disturbance of Basedow's disease is in the cerebral centers, especially those which control the nutrition of the thyroid and regulate the circulation. In cases where the disease exists there is an almost uniform evidence of neuropathic personal and family history, showing that these centers are constitutionally weak and this weakness is increased by emotional strain and infection. Fevers, sepsis, operation procedures and pregnancy are often followed by this disease, exophthalmic goiter more often following typhoid than the other infectious fevers. The author reports two autopsies after a rather acute course where pathological changes were found in the nerve-cells of the pons and medulla, which leads him to believe that in well-marked cases these changes in the nuclei of the pneumogastric and other nerves are constant and important lesions.

W. B. NOYES.

STUDIES ON NEUROGLIA. G. CARL HUBER (Amer. Jour. Anat., Vol. 1, No. 1).

From studies of the neuroglia of the dog, the cat, the rabbit, the dove, the tortoise and the frog this author concludes that the neuroglia of the spinal cords of these animals is made up of the neuroglia cells and fibers, the protoplasm of the fibers differing chemically from that of the cells, although in all the animals studied this difference was not equally marked. The fibers may be regarded as intercellular in structure, bearing no constant relation to the great majority of cell nuclei or neuroglia cells observed. Using Benda's method, the author has demonstrated that there are some neuroglia cells, usually possessing protoplas-

mic branches, in which the neuroglic fibers are not completely separated from the protoplasm, but are in continuity with it, or even passing through it. The fibers usually follow the course of the protoplasmic branches of the cells. JELLIFFE.

TETANY. SANGER BROWN (Med. News, July 5, 1902).

In the discussion of this subject this author mentions as frequent causes of tetany, thyroid deprivation, due to disease or surgical interference, gastrointestinal disorders, such as diarrhea and constipation, sometimes acute infections, or in women pregnancy and lactation. Sometimes hysteria and epilepsy are found in connection with tetany. Young adults are most subject to this affection and as yet no post-mortem examination has led to any clear comprehension of the nature of the disease. It is always marked by the occurrence of spasms, which usually decrease during sleep. These spasms are nearly always symmetrical, but variable in distribution, and the electrical excitation of both nerves and muscles is greatly increased. Such sensory phenomena as tingling or burning may be present, as well as vasomotor and trophic phenomena, such as edema of the parts of the location of the spasm, as cloudiness of the lens has been observed in some instances. The diagnosis is not difficult in typical cases. The duration of the disease varies from a few hours to months. With the exception of thyroid and pregnancy cases no specific has yet been discovered, and in cases resulting from extirpation or atrophy of the thyroid the treatment is obvious. The presence of a physician is necessary when it is imperative to employ sedatives and antispasmodics sufficient to subdue any violent paroxysm. The general characteristics of the disease are tonic spasms, either paroxysmal or continuous, and ordinarily symmetrical. These spasms are generally limited to the hands and feet, but in severe cases may be more extended. When sensory and general symptoms are present they are almost invariably distinctly subordinate. W. B. NOYES.

FATIGUE IN NON-MEDULLATED NERVES. T. G. BRODIE AND W. D. HALLIBURTON (Jour. Physiol., May 28, 1902).

It has been demonstrated by Waller that while nerve activity is going on there is an increase in the action current, somewhat like that caused by small doses of carbolic acid, and by this he infers that this substance is produced during the passage of the nerve impulse, resulting from the destructive changes in the substances which make up the fatty sheath. He is of the opinion that the gray axis in action both lays down and uses up its own fatty sheath, the process of nutrition following destruction so quickly that the evidence of fatigue cannot be demonstrated. These authors, assuming this to be the case, showed that it would be possible to show evidence of fatigue in non-medullated fibers, and those of the spleen were selected for experiment. The result of a stimulation of these fibers by an electric current was a contraction of the spleen, the volume of which was measured by an oncometer. Water, slightly above the freezing point, was passed through a tube, placed between the electrodes and the spleen, with the nerve resting upon it. The cold prevents the nerve impulses from entering the spleen, acting as a block. After the nerve had been stimulated for many hours the block was removed. When the nerve was again stimulated the contraction of the spleen was quite as vigorous as before the long-continued stimulation, which showed that the non-medullated nerve fibers of the spleen evinced no apparent evidences of fatigue. The authors believe that the "stimulation fatigue" referred to by Howell and others, is the result of polarization of the nerve after prolonged faradization and that an extended

rest must not necessarily precede re-stimulation. Although peripheral fatigue of the nerve-terminals may be readily induced in the spleen it does not nullify the results of these experiments. Stimulative fatigue can be induced in vasomotor nerves, but not in the nerves of the spleen. The transmission of nerve impulses is not barred by stimulation fatigue.

JELLIFFE.

PLEA FOR SIMPLER MEDICINAL TREATMENT OF CHRONIC NERVOUS DISEASE. JOSEPH COLLINS (Med. News, July 5, 1902).

This writer believes in the proper use of medicine, but calls attention to its frequent abuse. He is of the opinion that in many cases, such as neurasthenia, drugs should only be used to fulfil some pointed indication and substitutes for them exercise, rest, diet, conformation to the rules of health, avoidance of deleterious influences, with massage and hydrotherapy. Epilepsy is best treated by avoiding those experiences which have been shown to precipitate attacks, and putting the system in such a condition that the excitants of epileptic seizures, such as fatigue, autointoxications from the gastrointestinal tract and poisonous products, may be stayed by dieting, attention to the best hygenic measures, massage, suspension, electricity and hydrotherapy, and the patient should be assured that by attention to these measures the approach of incapacity which must eventually overtake him may be retarded for an extended period.

W. B. NOYES.

NEUROSES AS SEEN IN ORTHOPEDIC PRACTICE. B. E. MCKENZIE, M.D. (N. Y. Med. Jour., July 13, 1902).

Orthopedic practice includes many neuroses of the spine, where no changes in the central or peripheral nerve structures can be discovered. The neurosis may not be the only, and possibly not the chief disease present. After treating any abnormal condition the writer finds that discipline which educates the patient to be self-reliant must be enforced, and systematic gymnastic exercise carried on. The writer's cases present a large variety of these conditions. *Case I*, complaining of pain in the back and head, and inability to exert herself for many months, was cured by isolation from her family and systematic gymnastics. *Case II*, suffering for several years from backache, and symptoms of hip disease, was cured in a short time by gymnasium training, the entire trouble being hysteria. Other cases presented symptoms resembling Pott's disease, multiple neuritis with severe contractures, lateral curvature of the spine with great weakness, joint disease of various sorts. They all recovered under treatment isolating them from their families and instituting carefully selected physical exercises in a gymnasium under a competent instructor, preferably in classes. This treatment is a substitution or modification of the Weir Mitchell system of treatment.

W. B. NOYES.

THE CHANGES IN THE SPINAL CORD AND MEDULLA IN PERNICIOUS ANEMIA. FRANK BILLINGS, M.D. (Boston Med. and Surg. Jour., Aug. 28, 1902).

The writer discusses the characteristic changes in the spinal cord in pernicious anemia, and their resemblance to other conditions of diffuse degeneration. The cause of pernicious anemia is inferred to be toxemia, occurring after pregnancy, syphilis, malaria and fevers, as a rule associated with disorders of the digestive tract. The symptoms are chiefly due to the profound anemia. There are present nervous phenomena, usually subjective, and paresthesia, cerebral disturbance, fixation of the pupils and other nervous phenomena are observed. Spinal cord lesions,

occurring in a small percentage of the cases may appear as one of the earliest manifestations of the disease. Of the author's thirty-six cases paresthesia was noted early in the history, appearing simultaneously in feet and hands. Functional nervous disorder was noted in twenty-four cases. In ten cases a spastic and usually an ataxic condition more or less progressive was noted. In three cases complete flaccid paraplegia with loss of knee-jerk and voluntary bowel and bladder control appeared. Girdle sensation was present in the spastic cases contrary to the usual teaching. In three cases there were pathological changes in the cord with a slight sclerosis or degeneration in the lateral and posterior columns of varying intensity and location. There was no marked degeneration of the gray matter of the root zone, of the root ganglia, and of the brain cortex. The degeneration varied at different heights of the cord. It appeared in one case as a focal degeneration, varying decidedly at different heights, not involving a whole system centrifugally or centripetally. It is apt to diffuse itself from one tract to another, but involves the spinal gray as a rule. The whole nerve is not involved. There are two kinds of degenerative processes—an acute, where the nerve sheath becomes swollen, stretched and filled with debris, but not usually associated with an increase of the neuroglia. These lesions of the spinal cord have been attributed to multiple hemorrhages, thickening of the walls of the blood vessels, acute myelitis, primary changes in the gray matter, to anemia, or to a toxic agent. The writer's conclusions are as follows: (1) That there is a well-established relation of diffuse cord degeneration with pernicious anemia. (2) It seems highly probable that the hemolysis and the cord changes are due to the same toxin. (3) While the source of the toxin is unknown the fact that gastro-intestinal disturbance is so common in the disease would lead one to suppose that it is intestinal in origin. (4) The diffuse degeneration of the spinal cord which occurs in conditions without pernicious anemia, does not appear to differ essentially from those of pernicious anemia. (5) It is possible that a common toxin exists as a blood circulating poison in one individual, acts upon the nervous apparatus in another, and coincidentally upon the blood and spinal cord in others. W. B. Noyes.

The Nerves of the Heart. Horace H. Hoppe, M.D. (Jour. Amer. Med. Asso., May 24, 1902).

A nervous affection of the heart is caused by a faulty condition of the nervous system locally in the heart, the nerves leading to the heart, their center in the medulla, or by a derangement of the nervous system as a whole, as in neurasthenia and hysteria. While the heart is rich in sensory fibers and ganglionic cells, there are no motor fibers nor motor centers. The teaching of the action of the vagus and sympathetic has been modified. The motor stimulus comes from the blood itself. The rhythm of the heart action is automatic, but it can be diminished by influences, chemical, physical or psychical in character, coming either through the sympathetic nerve, or from the vagus. The three kinds of neurosis of the heart are: (1) neurasthenia cardis; (2) tachycardia; and (3) bradycardia. Of neurasthenia cardis we have (a) typical neurasthenia of the heart; (b) arhythmia of the heart; (c) pseudo-angina pectoris; (d) nervous bruit. The first has besides ordinary neurasthenic symptoms, cardiac hypochondria or fear of heart disease based on local precordial sensations, produced by palpitation, irregularities or pain. Fear is a predominent psychic element here as in other forms of neurasthenia. Palpitation is frequent, due to chemical or mechanical conditions of the stomach. It is often associated with a severe sense of constriction of the throat, or dysphonia. The typical neurasthenic heart is

weak, irritable and easily exhausted. Dilatation may easily occur. Arhythmia cordis is a condition where irregular or intermittent rhythm occurs even in the absence of all excitement. The most frequent cause is a diseased condition of the coronary arteries or myocarditis. It is not common in neurasthenia with marked vasomotor disturbance, and varies with physical or mental excitement. The nervous ganglia and nerves of the heart being purely sensory, may produce besides disturbances of sensation, distinct pain, either in paroxysms or more or less constant. This is a nervous angina pectoris. Coronary disease is often absent. In most cases the pseudo angina is a neuralgia and entirely functional. It occurs independent of effort, often in the night, associated with other signs of neurasthenia. Tachycardia may occur as a pure neurosis, independent of all signs of dilatation or other disease of the heart. It may be due to irritation of the sympathetic. Bradycardia is rare as a pure neurosis; it may be reflex from gastro-intestinal disturbances, and occurs in paroxysms. It is best treated by sedatives, such as bromides, asafetida and valerian. W. B. Noyes (New York).

Acute Ascending Paralysis in Basedow's Disease. M. Rosenfeld (Berlin. klin. Woch., June 9, 1902).

Regarding paralysis of the extremities occurring in Basedow's disease, to which Charcot and Mackinsie called attention, as occurring without loss of sensation, weakness of the bladder or muscular atrophy, this author relates the history of a case where the cachectic period of the disease began with an acute ascending paralysis, following a course similar to that of Landry's disease. Although the patient had been ill for two years, suffering during one year with exophthalmos and extreme nervousness, he had continued at work without interruption. On the day of the attack he experienced nausea and headache in the morning and later in the day lumbar pains and a feeling of weight in the lower extremities. The next day pain and weakness compelled the patient to stop work, but there was no fever and the cranial nerves and sensation were normal, as well as the movements of the upper extremities, although walking or standing was impossible. The right leg was absolutely powerless and the patellar reflex absent, but the left leg could be moved a little and a slight reflex obtained on that side. The bladder and rectum remained normal and the chief complaint was of lumbar pain radiating to the extremities. After a few hours the power of sitting erect was lost, while the arms could only be moved with difficulty, and before night there was complete paralysis of the arms and the head could not be moved. There was slight motion in the fingers but the bladder was paralyzed, although respiration continued normal without loss of consciousness. The next day voluntary motion of the extremities returned but a rapid loss of weight ensued, together with an increase of all the usual symptoms of Basedow's disease and a permanent loss of muscular power. This attack was followed by two others which were neither of them as severe as the first. Hysteria may be excluded on the following grounds; the prodromata, the severe collapse at the outset of the paralytic attack, the paralysis gradually ascending, the disappearance of the right patellar reflex during the paroxysm and the existence of a light paresis of the right leg with diminished reflex after the attack.

Jelliffe.

Book Reviews

Anatomie des Centres Nerveux. Par J. Dejerine et Madame Dejerine-Klumpke. Tome deuxième. Fascicule 1. Avec 465 figures dans le text dont 180 en couleurs. Paris, Rueff, 1901. Price 32 frcs.

The work which I reviewed over six years ago in this Journal, has found its continuation. The first half of the second volume lies before us, containing as second part the chapters on the projection fibers of the cerebral cortex-mantle and rhinencephalon (p. 1-304), the infracortical ganglia, corpus striatum, optic thalamus and subthalamic region (p. 305-411), and the cranial nerves derived from the forebrain and interbrain—the olfactory and the optic (p. 412-435). The third part deals with the rhombencephalon; the first chapter with its general morphology (p. 436-503), the second with its inner configuration (p. 504-586), the third with a description of serial sections (p. 587-690), and the fourth with the finer texture (p. 691-720).

Like its predecessor, the volume is gorgeously illustrated with fine original drawings usually representing entire series, or in the case of the numerous pathological brains with secondary degenerations, at least the most important levels. Taken altogether, the volume is a representation of the richest collection of studies of secondary degeneration ever made the foundation of a monograph. Fifty-three cases underlie the descriptions and conclusions concerning the cortico-thalmic connections. In some of them, it is true, one would have liked the entire material presented to make it more conclusive, as, for instance, in case Prudel p. 101 the depth of the hemisphere-lesion to explain the question of decay of the pulvinar in view of the integrity of the calcarine area and fusiform and lingual gyri, since very important conclusions are built on other facts in this case. Besides the anatomical facts, there is a very good summary of the chief data of localization of function in the cortex and in the internal capsule. In the latter, D. especially insists on the error of Charcot of attributing a complete hemianesthesia to the "carrefour sensitive," the posterior end of the internal capsule. What Charcot described was hysterical hemianesthesia (including smell, taste, hearing and amblyopia, not merely hemianesthesia). Capsular hemianesthesia is either due to simultaneous lesion of the thalamus termination of the fillet and the corresponding thalamo-cortical radiation, or to lesion of the thalamo-cortical connections to the kinesthetic area of the cortex.

Among the more important anatomical statements the following may find a place in this brief review:

The crus cerebri contains no fibers from the corpus striatum; the inner portion comes, not from the entire frontal lobe, but from the central operculum and the foot (insertion) of the 3d frontal convolution; and the bundle of Türck comes from the middle portions chiefly of the second and third temporal gyrus, while the pyramidal tract occupies the middle three-fifths of the crus.

Among degenerations, D. points out the existence of cellulifugal and cellulipetal degenerations, and indirect or secondary atrophies, and further "atrophies en masse" of the homolateral half of the brain-stem

and the crossed side of the cord, and compensatory hypertrophy of the healthy side of the crus and pyramids. With the Marchi method cellulifugal degeneration is visible before the cellulipetal decay which "rapidly diminishes in intensity as one gets away from the lesion" (at variance with Bregmann).

The connections of the rhinencephalon, fornix, and corpus striatum and thalamus receive a specially full description and illustration. The fasciculus occipito-frontalis is maintained as a real occipito-frontal association-path. The globus pallidus and corpus Luysii are shown to have some connection with the cortex. The ansa lenticularis receives a new description at variance with that of Edinger and Flechsig, on ground of Marchi specimens. The existence of Gudden's commissure in man is denied. The description of the thalamus and its connections are very carefully done.

The rest of the volume contains the well illustrated systematic description of serial sections of the hind-brain. The second part, including the anatomy of the spinal cord, is in press.

The second volume greatly surpasses the first one in a wealth of new observations and demonstrations of pathological anatomy, and makes us wish that the authors may be in a position to soon finish the work, which, together with the "Sémiologie" is a wonderful monument of the co-operation of a brilliant French scholar and his equally brilliant American wife. A. MEYER.

TRAITÉ DES MALADIES DE LA MOÊLLE ÉPINIÈRE. By J. DEJERINE and ANDRÉ THOMAS. J. B. Baillière et Fils, Paris, 1902.

One may well be surprised that Dejerine's name should be found as the joint author of a book on clinical neurology so soon after the appearance of his "Sémiologie du système nerveux," but in the work published in collaboration with André Thomas the subject is presented in the more customary manner, in that each disease of the spinal cord is treated as an entity, and the viewpoint is not one of symptomatology. Syphilis of the spinal cord is omitted because the article by Dejerine and Thomas forms a part of Brouardel and Gilbert's "Traité de mèdecine et de thèrapeutique," in which syphilis is considered by other writers.

In Dejerine and Thomas' book the plan of illustrating adopted in the "Sémiologie" has been followed, and the numerous pictures are presented with full explanatory legends, so that much information can be gained by a careful examination of the illustrations and their accompanying legends.

A brief consideration of anatomy, physiology, pathology and general symptomatology opens the way for a presentation of the individual diseases. The latter are divided into the secondary affections of the spinal cord (compression, Pott's disease) and into the primary affections of the cord (myelitis, tabes, etc.).

In many places where disputed questions are discussed the views of different authors are fairly given, occasionally without any attempt to determine which views are correct. This is doubtless wise, because a presentation of facts where a positive conclusion is impossible, is all that can be expected. In other places, however, Dejerine and Thomas state their own convictions clearly.

Under the title of Little's disease a historical account of the affection is given, and the limitation placed by certain authors in their description of the symptom-complex is mentioned. The views of most of the prominent writers on this much-disputed subject are clearly set forth, so that the chapter contains almost all that is now known regarding Lit-

tle's disease—a term not very frequently employed in America, although by no means unknown. Dejerine has shown by a case with necropsy that Little's disease, or "congenital spastic rigidity of the limbs," may be purely of spinal origin, and Dejerine and Thomas therefore believe that the symptom-complex of Little in some cases may be caused by cerebral lesions, in others by spinal lesions, and they hold that cases in which the symptoms are capable of amelioration from year to year should be distinguished from the unimprovable ones—a view which certainly seems proper.

In regard to Landry's paralysis Dejerine and Thomas say that it is an affection of the peripheral motor neurones, and according as the cell-bodies of origin or the axis-cylinder processes are implicated the malady is either poliomyelitis or polyneuritis. The acute ascending paralysis is, however, only a symptom-complex.

Chronic anterior poliomyelitis in their opinion exists, although Marie has sought to exclude the disease. They acknowledge that Duchenne included under the name of progressive muscular atrophy such affections as syringomyelia, amyotrophic lateral sclerosis, myopathy and peripheral neuritis, but they assert that the symptom-complex of Duchenne may result from atrophy of the cell-bodies of the anterior horns without alteration of the pyramidal tracts. They do not accept Leyden's views that amyotrophic lateral sclerosis is to be classed with the muscular atrophy of the Aran-Duchenne type, or else with the labio-glosso-laryngeal paralysis of Duchenne;—on the contrary they regard amyotrophic lateral sclerosis as a distinct disease.

It is not necessary to add that this book by Dejerine and Thomas will prove valuable to all interested in nervous diseases.

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Original Articles.

REMARKS ON ACUTE MYELITIS, AND REPORT OF A CASE OF TUBERCULOUS MENINGO-MYELITIS.

By Joseph Collins, M.D., New York.

(from the pathological laboratory of the new york state hospitals, directorship of dr. ira van gieson.)

The question whether acute myelitis is a rare disease or not may be answered in the affirmative or the negative, depending entirely upon what is included under such a designation. If we include acute transverse myelitis, acute diffuse myelitis ascending and descending, acute disseminated myelitis, acute anterior poliomyelitis, as Leyden and Goldscheider do, it is the commonest disease of the central nervous system, for it takes in diseases which are well recognized as either pathological or clinical entities, such as infantile paralysis, Landry's paralysis and acute hemorrhagic softening associated with disease of the blood and the vessels. It includes also the destructive lesions accompanying trauma, diminution of atmospheric pressure (caisson disease), and disease of the abdominal aorta leading to occlusion, and obliteration of the vessels leading to softening. From the clinical standpoint these cases are truly myelitis. But if we make circulatory manifestations of inflammation the criterion by which to judge whether or not there is any inflammation, many of these conditions must be excluded. For instance, softening resulting from trauma, obliteration of the vessels, diseases of the blood such as anemia, edema, inflammatory or other-

wise. From a pathological standpoint, however, such conditions are to be differentiated by the absence of changes in the circulation typical of inflammation. If they are differentiated, and not included under the caption of acute myelitis, then the disease is extremely rare. If they are included—and they are usually clinically, for they can not be distinguished by the clinical picture alone, then the disease is, as has just been said, not uncommon. Whether or not it is a common disease differs also with our conception of acute myelitis from an etiological standpoint.

If we consider acute myelitis to be an inflammatory state of the spinal cord due to infection or intoxication, the morbid process being caused either directly by the action of the microbes and their toxins acting upon the medullary parenchyma, or indirectly through the circulatory system, then the disease is really very rare. As a matter of fact, primary acute myelitis (leaving out of consideration anterior poliomyelitis), whether it be transverse, diffuse or disseminated, is a rare disease and nearly always due to infections or intoxications.

The infectious organisms that produce such myelitis are numerous. Those that have been found in the inflammatory focus are the streptococcus, the staphylococcus (at least two varieties) the pneumococcus, and a special diplococcus described by Buzzard and Russell. In the case described herewith, one of acute meningo-myelitis, the tubercle bacillus was found in the inflammatory focus.

Mere enumeration of the acute diseases that have been closely followed by myelitis, and with which it is safe to assume the myelitis stands in causal relationship, shows that the number of organisms capable of causing myelitis is not exhausted by those above mentioned. Thus it has been shown that myelitis follows tonsilitis, dysentery, erysipelas, typhoid fever, gonorrhea, cystitis, puerperal fever, pneumonia, influenza, rabies, tetanus, phlegmonous paronychia; to mention no others. Undoubtedly, in many instances the germ itself does not produce the disease, but the toxins which they manufacture cause it. It is not safe however, to assume that the germ does not cause the disease *per se,* and by its local action, because it is not found in the focus after death, for it is well known

that the germ begins to disintegrate and disappear the moment it has exercised its malign effect. The fact that the germ is sometimes found on microscopical examination, as it was in the case reported herewith, shows that at least some of the cases of acute inflammation of the cord are dependent upon the immediate presence of the organism itself.

The patient whose history I present was admitted to the City Hospital April 19, 1898. She was a Polish Jewess, twenty years old. So far as could be learned, her family history was negative. Her personal history was likewise uninteresting: a hard-working, underfed corset-maker, who denied the use of spirits and in whom there were no evidences of venereal infection. When a child she had measles, but since then she had been entirely free from illness. Her first child was born in the hospital two months after the onset of the illness which caused the patient's death. This illness began about three months before its fatal termination. She said that the first manifestation of the disease was that one morning on arising the right hand "was sore;" which being interpreted means that the second metacarpo-phalangeal articulation was red and swollen, and that the finger could not be moved because of the swelling and of the pain which attempts at movement caused. Even when the extremity was at rest, this joint was the seat of sharp, stinging pain. This pain continued for two or three days; then the right elbow became painful and somewhat swollen. The pain and swelling subsided slowly, and after a day or two the right shoulder became affected in a similar way. In two or three days the swelling and pain of the shoulder began to subside, and then she complained of pain in the middle of the back. The pain and stiffness of the back were not so unbearable as the pain in the other joints. Moreover, they disappeared in a few days. The anterior surface of the right thigh and the right groin then got painful. Later, the right knee became stiff and swollen, and she soon was unable to walk or take care of herself. She was then taken to Bellevue Hospital and remained there for three weeks under treatment for rheumatism, she said. The record of her case while in that institution has not been seen, but when she was transferred to the City Hospital from Bellevue it was noted there was a bedsore over the sacrum, one over each iliac crest, one over the anterior superior spinous process, one over the left trochanter, and one over the right knee. All these sores were extremely painful, especially that on the back. From all of them pus discharged freely. She re-

mained in the ward to which she was at first admitted for a short time only, for it soon became apparent that she was about to give birth. She was transferred to the Maternity Hospital, and was delivered of a child that is now alive. She had no labor pains, but during the birth she complained of great pain in the joints, due apparently to inability to separate the legs and to remain on the back. A few days after her confinement she was sent to one of my wards, where the following notes were made:

"The patient who is extremely emaciated, lies on the back, the feet and toes semi-flexed, and the knees and thighs firmly flexed. When attempt is made to overcome the flexion the patient complains bitterly. The legs and thighs are very much wasted. [This was not considered other than part of the general emaciation, although it is very probable that it was likewise due to disease of the ventral horns which was afterwards found.] The knee-jerks can not be elicited, nor can the spasticity of the legs be overcome. The absolute fixation of the lower extremities is thought to be due in great measure to involuntary efforts at restraint to avoid the pain that movement causes. The patient is wholly unable to move any part of the lower extremities, but she complains of painful twitchings of the muscles and of uncontrollable drawing up of the legs. She is incontinent of urine, most of the time, but the rectal sphincter seems to functionate normally. There are no elicitable disturbances of sensibility. [It was difficult to be certain of this on account of her fear that she was going to be hurt by the examination.] The sore on the back, over the sacrum, is as large as two hands and it has destroyed the soft tissues down to the bone. The finger or probe can be admitted for a distance of from three to four inches. This procedure is very painful, causing liberal discharge of pus, just as if a new well had been opened up. The other bedsores enumerated above are of the extremest kind. The upper extremities are emaciated and slightly rigid, but not devoid of voluntary power. There is no deformity of the spine. The cranial nerves are unaffected, and the patient gives a lucid account of her illness." Careful examination of the thoracic and abdominal viscera failed to reveal anything pathological. When first admitted to the hospital, the records show that she was without fever. About two weeks before death she began to have rigors and irregular elevation of temperature, and to show the customary phenomena of septicemia.

Autopsy eight hours after death. The body is in the same fixed position, especially the lower extremities, that it was *ante mortem*. The bedsore of the posterior surface of the sacrum

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Fig. 4—Swollen homogeneous ventral horn cells. Beginning coagulation of the achromatic substance.

Fig. 3—Second lumbar s
showing varying grades of ch

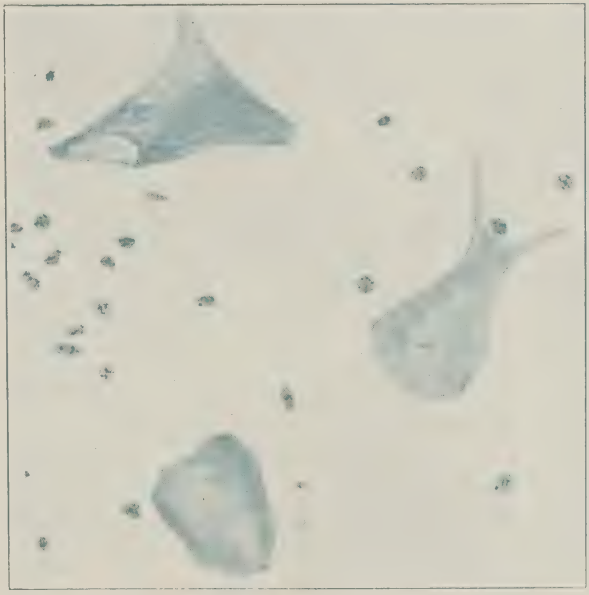

Fig. 5—Fifth lumbar. To show swollen condition of anterior horn cells and homogeneity.

Fig. 2—Second lumbar segment, showing paucity of cells in anterior horns and initiation of degenerative stage in cell substance. Disappearance and dislocation of nuclei.

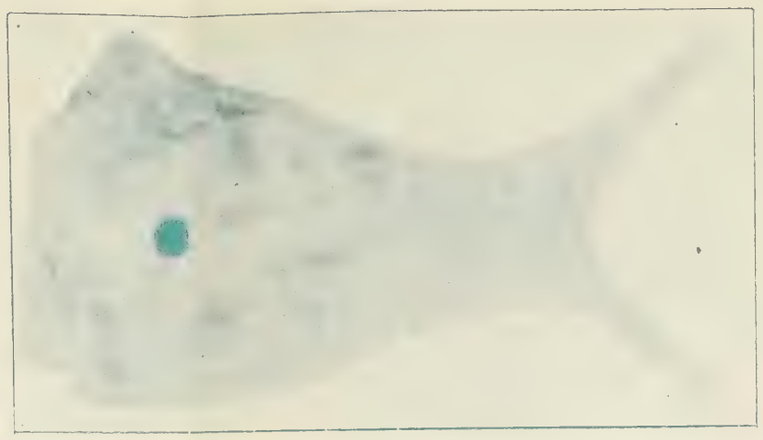

Fig. 6—Enlargement of colorless space around nucleus and partial disappearance of tiger-hide appearance (high magnification).

Fig. 4—Swollen homogeneous ventral horn cells. Beginning coagulation of the achromatic substance.

Fig. 3—Second lumbar segment. Anterior horn cells showing varying grades of chromatolysis.

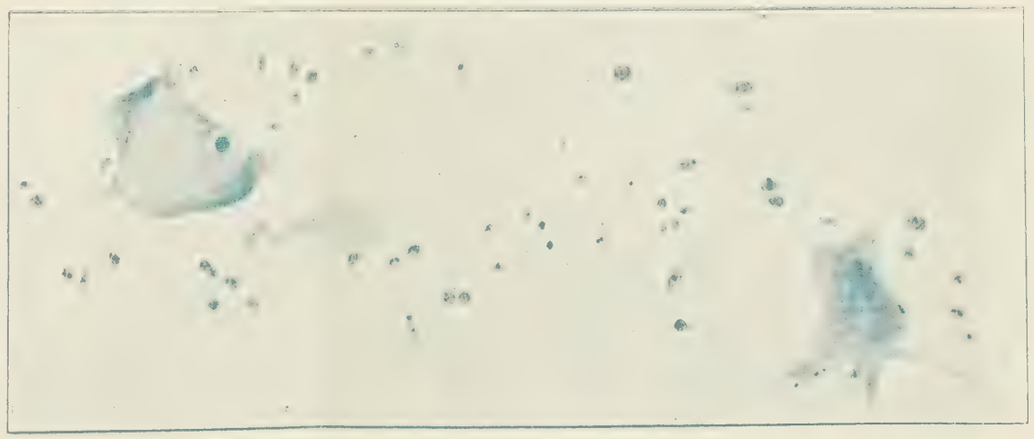

Fig. 7—Fifth lumbar. Showing partial chromatolysis and advanced chromatolysis and dislocation of nucleus due to destruction of the achromatic reticulum.

Fig. 5—Fifth lumbar. To show swollen condition of anterior horn cells and homogeneity.

exposes the bone. On opening the spine, the canal is lined with pus up to the second lumbar vertebra. From this point upward between the pia and dura is a freshly organized layer of greenish yellow exudate, apparently made up of fibrin, pus, and coagulated serum. This layer reaches up through the foramen magnum along the base of the brain and over the optic chiasm. The brain itself is apparently normal. The consistency of the cord is diminished throughout, and from the sixth to the ninth dorsal segments it is so soft that it flows on being cut. The cord was divided into segments and every other one hardened in alcohol and Müller's fluid respectively, preparatory to methylene blue, hematoxylin and carmine stains. Examination of the viscera showed dilatation of the heart, both the right and left sides, and slight fatty degeneration. The lungs were free from tuberculosis. The kidneys were in a state of interstitial and parenchymatous degeneration. No tuberculous foci were found in the thoracic or abdominal viscera.

The changes in the spinal cord on microscopical examination were of variable intensity and distribution. They were most marked in the eighth and ninth dorsal segments, wherein they constituted a focus of central myelitic destruction, surrounded by an annular area of myelomalacia, and were least marked in the cervical cord wherein they consisted of slight perivasculitis, proliferation of neuroglia nuclei, and increased connective tissue formation, both from pial prolongations and from the vessels. The changes in the lumbar segments were most conspicuously evidenced by changes in the substance of the parenchyma. From one end of the cord to the other, the dura was the seat of profound productive inflammation, particularly that portion of it surrounding the posterior segment. The pia was likewise the seat of an exudative and productive inflammation, but it was not implicated so profoundly as the dura.

A number of sections from the different levels of the cord will now be considered in detail, and as the lesion was most intense in the dorsal cord, I shall describe first the changes at this level. The sections made from the eighth dorsal segment and stained according to Weigert and Pal's method show a central area of necrosis, limited in front by the commissure which in some sections is partly, and in others almost wholly destroyed. It is probable that this complete destruction is artificial, as it was very difficult to cut and handle these sections, on account of the destructive changes that had gone on in them during life. Posteriorly, the punched-out area extends about one-third of the distance to the posterior border of the cord. The margin of the focus is made up of detritus and beyond this

is an area varying in each section, made up of enlarged and thickened blood vessels, and proliferation of connective tissue with conglomeration of round cells in the perivascular spaces. The pia is very much thickened, especially on the posterior and lateral surfaces of the cord, and sends comparatively few prolongations into the substance. The enormous exudative and proliferative change on the free surface of the pia is very conspicuous in this section. One of the sections from this level stained according to the method of Zeihl, revealed the presence of the bacillus of tuberculosis in the center of a bit of homogeneous material at the margin of the focus of destructive myelitis.

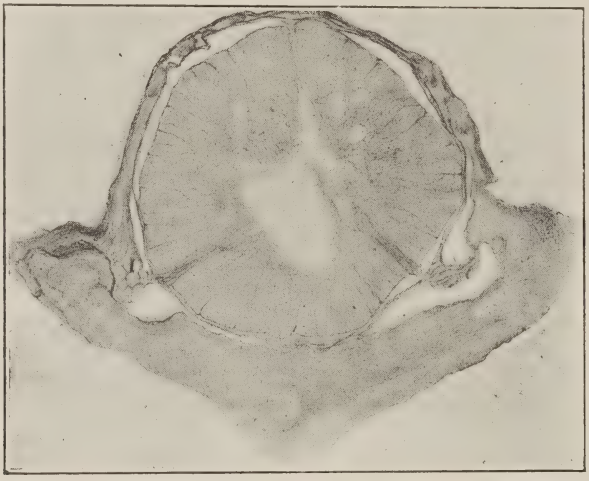

Fig. 1—Eighth dorsal, showing central myelitic focus and a colossal meningeal involvement.

This section was given to the artist to be drawn, but it has been misplaced. The existence of the bacillus was corroborated by Dr. Onuf, by Dr. Harlow Brooks, and others of the staff at the Pathological Institute. No bacilli were found in the meningeal exudate, although many specimens were stained with this end in view. Sections from the lower dorsal levels stained with methylene blue show that the changes in the ganglion cells are those of advanced diffuse chromatolysis. (Here it may be said that the term chromatolysis is used only to mean disintegration of the chromatic particles of the protoplasm.) In some of the cells this chromatolysis is principally peripheral, but in the most it is perinuclear or diffuse. The achromatic part has suffered as well. In many of the cells there is complete disappearance of

the achromatic fibrillæ, while in others there is absolute plasmolysis. The cells that were particularly affected at these levels were those of the internal groups of the anterior horns. It is now generally accepted that chromatolysis is the reaction of the cell to a disturbing force and is reparable, while alteration of the achromatic part is degenerative and irreparable. This view is entirely in keeping with the findings in the dorsal segments in which the cell-bodies have lost not only the chromophile but have undergone plasmolysis. The changes of the cells in the upper dorsal and cervical cord are in a measure those that would occur in every spinal cord that had been subject to injurious influences.

Sections from the twelfth dorsal segment, stained according to Weigert, show complete disappearance of the central area of myelitis, but this part of the cord has the appearance of being brittle, and was difficult to section without breaking. Sections from the first lumbar segment stained with Van Gieson's picro-carmine, show thickening of the meninges and the inflammatory exudate. The most noteworthy revelation of this stain, however, was the condition of the blood vessels of the anterior horns; these vessels were seen to be in a state of congestion with resulting transudation and diapedesis, which in part at least conditioned the changes in the ganglion cells of the cord revealed by Nissl's method, which I am about to describe. The degeneration of the white matter of the cord caused by the accompanying meningitis, and revealed by Pal's method, is particularly evident in this and in lower levels of the lumbar cord. Lissauer's column suffered most severely. The central posterior columns were so brittle from the extension of inflammation into them that it was scarcely possible to cut a section without crumbling this part of the cord.

Sections from the various lumbar segments stained with the Nissl stain showed a paucity of cells and a variable degree of degeneration and disintegration of the cell-bodies and processes of the anterior horn. From three to five cell-bodies of each anterior horn were the seat of well-marked degeneration. In some this is so extensive that there is no longer any similarity to normal in the cell-bodies affected, while in others the degeneration is extremely variable. Some of the largest cell-bodies had apparently undergone complete homogeneous transformation, while others show at the periphery narrow marginal lines made up of dots which take a deep blue color, evidently the remains of chromophilic granules. The processes of the cell-bodies show a degeneration very similar to that of the bodies. For instance, some of the processes have the appearance merely of a colorless

thread whose continuity, however, can be distinctly traced, but only in one or two places throughout its entire length is there anything like a color dot or spot. Other processes are degenerated at the terminal ends, while the part in connection with the cell-body itself still retains some of the normal longitudinal striation. The cell-bodies which are only partially degenerated preserve to some extent the normal tiger-hide appearance. They have merely undergone some homogeneous transformation which renders them non-receptive to the Nissl stain. The cell-bodies that are least affected show merely an enlargement of the colorless space which normally surrounds the nuclei, and a partial disappearance of the tiger-hide and dotted appearance. The changes in the nuclei of these cell-bodies are variable. In some the nucleus has disappeared, in others it has changed its location from the center of the cell-body, where it is ordinarily found, to the periphery.

The changes in the cell-bodies of the anterior horns of the cervical spinal cord are, especially in the lower segments, very similar to those in the lumbar region, though less pronounced. In the upper cervical cord the structure of the cell-bodies constituting the different groups is not materially altered, despite the fact that the blood vessels of the anterior horns are thickened, the peri-arterial spaces much enlarged, and the neuroglia very much increased. As we proceed downward in our examination of the sections through the cervical cord, we find various manifestations of pathological changes in the cells of the anterior horns. At first there is evidence that the chromophilic granules in many of the cells are large and stained very deeply, while the achromatic substance is quite normal.

Sections from the cervical cord stained with hematoxylin show most strikingly the enormously thickened pia which at these levels is implicated most profoundly over the anterior surface of the cord. This thickened pia extends down the anterior median fissure forcing it widely apart, especially at the bottom, where the pia lies in thick folds extending laterally beneath the columns of Türck, and thence sending prolongations which extend into the anterior horns, the anterior horns themselves being the seat of numerous blood vessels of enlarged caliber and thickened walls. The cell-bodies of the anterior horns from this level when stained with methylene blue show various stages of disintegration in the chromatic substance, with relative preservation of the achromatic substance. In a few sections a blood vessel of the anterior horns cut longitudinally is apparent, and in this diapedesis of the corpuscles can be readily seen. The walls of the blood vessels show very great thickening, especially the internal coat.

As we approach the dorsal region, the cell-bodies of the anterior horns show more marked degenerative changes and are very few in number. A remarkable feature of the cord at this level is the relative preservation of the cell components of Clarke's column. In other sections in which there is conspicuous vascular change in the pial proliferation, the cell-bodies of the anterior horns are relatively well preserved. That the inflammatory process in the meninges of the cervical cord was a relatively recent process, was shown by the fact that although many of the posterior roots at one level or another are completely surrounded and strangulated, as it were, by the thickened meninges, there is despite this very slight ascending degeneration in the posterior columns.

To summarize the clinical history and pathological findings in this case: a young married woman, four months pregnant, is taken ill without attributable cause, with symptoms of acute inflammatory rheumatism. After three weeks, the symptoms subside and are followed by such indications of acute myelitis as motor paralysis of the lower extremities, incontinence of urine, pain in the back and extremities, spasmodic twitchings and contractions of the legs, and enormous bedsores and other associated trophic disturbances. Soon after she gives birth to a living child, the labor being painless. From this time onward the symptoms of meningeal involvement which heretofore had not been conspicuous, become extremely evident. Later, the manifestations of sepsis precede death. On autopsy there is found an extensive purulent and productive leptomeningitis and inflammation of the spinal cord, particularly of the lower dorsal segment. Microscopical examination of this area shows that the focus of most extensive myelitis, consisting of absolute destruction of an area of the central gray matter surrounded by an annular area of myelomalacia, is confined to the seventh, eighth and ninth dorsal segments. The pathological changes in the cellular elements of the cord at these levels are those of extensive, diffuse chromatolysis and plasmolysis, while the changes in the vascular tissues are those of infective inflammation; and those in the white matter of the cord, those of simple inflammation and production of connective tissue. In one section of this level the bacillus of tuberculosis was found imbedded in a mass

of caseous necrotic tissue. In the lumbar and cervical segments where there were no evidences of infective inflammation, the cells of the gray matter were found in various stages of chromatolysis, the chromatic substance bearing the brunt of the lesion. Judging from the associated vascular involvement, and the production of neuroglia and connective tissue, the result of marked involvement of the meninges at these levels, these changes in the integral structure of the cell-bodies were the result of these pathological accompaniments as well as of the infectious process which had destroyed the cord at another level.

It seems to me that an interesting point for discussion is the relationship of the rheumatic attack, if rheumatism it really was, to the occurrence of the tuberculous myelitis. Furthermore, it would be interesting to know whether the changes in the cells of remote portions of the cord, constituting chromatolysis, were secondary to the tuberculous infection and the septicemia which immediately conditioned the patient's death, or whether they were the result of nutritional changes due to disease of the blood vessels and impairment of nutrition secondary to the extensive productive inflammation of the meninges.

Personally I am inclined to the view that the patient did not have acute inflammatory rheumatism, but that the pain in the extremities and swelling of the joints which were early symptoms were the result of tuberculous infection of the cord, which implicated in the beginning that part of the cord in which is situated the central representation of the sympathetic nervous system,[1] and that the entire early symptomatology can be explained on this supposition. The symptoms next to develop were the classical ones of slowly progressing acute myelitis, such as the tuberculous variety almost always is. Later, the symptoms of meningitis were superimposed, and they obscured somewhat those due to inflammation of the cord itself.

I reserve further discussion of the pathological features of this case until the publication in this Journal of another article on myelitis.

[1]See article by Onuf and Collins on this subject in the Archives of Neurology and Psychopathology, Vol. iii, Nos. 1 and 2.

REPORT OF A TRANSVERSE LESION OF THE MID-THORACIC SEGMENTS LEAVING INTACT THE POSTERIOR COLUMNS, AND CAUSING SYRINGOMYELIC DISSOCIATION[1].

By Adolf Meyer, M.D.,

director of the pathological institute, ward's island, new york.

(From the Worcester Insane Hospital.)

Gowers mentions a case of his own and one of Müller's in which definitely limited regions of the spinal cord were destroyed so as to produce a dissociation of the various sensory qualities. Their cases had the advantage of being unilateral or partially unilateral lesions throwing light on the question of homolateral and contralateral interference with sense qualities. Our own case is less advantageous since the lesion is fairly symmetrical, but it forms in many respects the negative of the case of Müller.

Gowers saw after destruction of the ventral two-thirds of one side of the third cervical segment and mere "swelling" of the posterior column entire loss of sensibility to pain on the opposite side without any impairment of tactile sensibility.

Müller, in a case in which exactly the parts were severed which were intact in Gowers' case (both posterior columns and the rest of one-half of the cord), found loss of tactile sense on both sides and loss of pain sense opposite to the lesion.

In our case there was a destruction of practically the entire cross-section of the fourth to sixth thoracic segment, leaving intact only the dorsal two-thirds of the posterior columns and a few pyramidal fibers. Clinically the case presented for probably two years before death:

Normal tactile sensibility, pressure sense, tickle sense and sense of position.

Analgesia and thermanesthesia below the sixth rib on the right and the fifth rib on the left.

A zone of slight thermo-hyperesthesia over the fourth rib.

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[1]Read at the annual meeting of the American Neurological Association, June 5, 6, and 7, 1902.

Girdle feeling (occasional) around the level of the umbilicus.

Paralysis below the thorax (allowing but a slight pull on the upper end of the rectus abdominis, and occasionally a slight pull on the thigh and also slight movement of the right toes).

Occasional involuntary pulling up of the thighs, at times accompanied by shooting pain.

The following is a summary of the clinical record of Dr. R. R. Gurley, and a statement of the anatomical findings (sections made by Dr. C. B. Dunlap).

The father of the patient died with general paralysis in a status epilepticus (intemperance and fast life).

Born 1857, of Protestant parentage, the patient went to school up to seventeen, and afterwards worked in a grocery store, and later in a large dry goods store of the West. He led a very fast life, both with regard to alcoholism and to venery, but did well in business. Several not further specified venereal infections.

In 1893 the patient began to feel "leg weary" at night, and at times would have an acute lameness in the legs, which would pass off. Winter 1893-94 one night his legs suddenly gave way, but the next day he was "as well as usual," and in a week resumed work, but was noticed to drag his left foot and frequently felt weakness and at times temporary numbness (it is not said whether motor or sensory). In the spring, 1896, he had influenza and double otitis. He came East in July devoid of ambition and interest, somewhat dull and dazed; he seemed to want to sleep all the time and walked with a couple of strong canes. After a short stay at the seashore he showed complete *defect of memory* of this outing, unless he was reminded of things, and he had much more difficulty in getting about. Since then he would often fall asleep in his chair for hours, snore vigorously and awake in a dazed condition. He began to fabricate, excused himself, thought the family had mixed him up. In the course of the winter he became more helpless. From February 1898 he required crutches, but still attended to the calls of nature. His memory seemed good for days previous to his coming East. All idea of time calculation since then was lost. He would not remember what he read, even if he had just laughed heartily over it.

Shooting pains began to appear combined with marked twitchings of the legs, which at times made sleep almost impossible. The legs would jerk upward, the left leg always higher and more briskly than the right, and fall back lifeless.

From March, 1898, he could not feel the passage of the feces. He began to soil the bed and finally preferred to sit on the stool all the time, because he forgot when he had had a passage, and continually asked for the stool again. A ring of inflammation, and finally decubitus formed on the buttocks, totally devoid of pain. The left leg was earlier affected, without power and seeming dead. There was also marked girdle pain—"a feeling of a bar of hot iron around the waist"; in April the legs felt like blocks of wood, except for occasional pains; the feet felt cold and the attendant had to rub them often. About April sixth he had the first trouble with micturition; "the water would come in drops." The patient became depressed, spoke at times of locomotor ataxia, was somewhat restless, at other times he was cheerful; the memory remained very poor. Friends whom he saw almost every day he claimed daily not to have seen for years. He claimed to have attended to business during the last two years, "had arrived in Boston a few days ago."

On admission, April 19, 1898, the patient appears with a bright, intelligent countenance and pleasant manner. He speaks without hesitation, and pursues an interesting and rational stream of thought beyond occasionally telling the same story twice or even four times within fifteen minutes, every time with the same pleasure. He soon learns to call the physicians by name and recognizes each separately, but will say: "Doctor, you haven't been near me for an age." He will read the same item in the paper over and over with apparent interest, does not know that he is taking medicine three times a day, has a faint remembrance of having gone to the seashore and a good memory of what happened before the last six months in the West, for instance, the buildings of the World's Fair, *etc.* But the memory of venereal history is very deficient. He usually forgets that he is in Worcester; also the time: "it is only three weeks since I was in Chicago." During conversation he often begs the physician's pardon very politely and says: "Will you please call my attendant? I am obliged to answer the calls of nature," and in a few minutes he forgets the "urgent necessity." The patient has insight into his physical condition, says he keeps up courage and hopes that he is getting better from day to day.

Physically he is very well developed and nourished. There is no scar on the penis. The pupils react promptly, the left ear is deaf (otitis); taste and smell are normal. Tactile sensibility is normal, also localization. A zone of *tactile hyperesthesia* on the right from the upper border of the sixth rib to a parallel about 8 cm. below and on the left from the upper border of the fifth rib about the same width downward. Feeling of girdle

sensation "like pants buttoned tight at the level of the umbilicus."

There is *analgesia* below the sixth rib on the right, and the fifth on the left. Also *thermanesthesia.* At the fourth rib there is apparently a slightly *hyperesthetic temperature zone.* Everywhere below sensations are given as "equally warm," but over the fourth rib, the cold tube is called "red hot."

The elbow and wrist jerks are equally increased on both sides, also the knee-jerks; the knee-cap reflex marked on the left, absent on the right. No ankle-clonus, plantar reflexes obtained only on sharp stimulation or not at all. At other times even a slight prick of the legs produces an involuntary retraction of the legs. The cremasteric reflex prompt on the left, absent on the right. *Tickle sense preserved.* On the outside of both legs the skin is hairless. There are no obvious atrophies. The muscular power in the head, back and arms is good everywhere, but there is a rather coarse tremor of the hands at rest and on rotation; no twitchings in the face. Below the thorax there is total paralysis. The patient only can flatten slightly the protuberant abdomen by a pull at the upper insertion of the recti. With the utmost effort he can pull up the thigh slightly, and at times move the right toes a little. Twitchings consist in a sudden contraction of the muscles of the thigh, an upward and inward movement of the leg, flexion of the thigh on the abdomen but no contraction of the muscles of the feet or calf or of the abdomen. When the knee reaches an angle of 60° it falls to a lifeless abduction and the leg extends itself again. Then comes a "hot iron pain" shooting through the leg from the toes to the top of the pelvis.

The status remained the same throughout the summer. On December 5, 1898, an ingrowing toe-nail was removed without anesthetic. The rubber cord used felt "very tight but not painful." During the operation he asked a dozen times at intervals of one or two minutes, "Have you taken the nail off, Doctor?" and finally claimed he had never asked.

July 20, 1899, the patient was oriented concerning the "Worcester Lunatic Hospital," claimed it was June 1897, or 1898, he knew the name of Dr. G., but not of other physicians, and that of only one attendant, and none of the patients. He thought he left the West six weeks ago. Simple calculations were fair; a striking contrast existed between absolute clearness of intellect, and memory defect; practically no fabrications.

Nov. 8, 1899, at 9 A.M., when the patient was given an enema he suddenly complained of a queer sensation, sickness of the stomach, severe pain in the shoulders, he vomited, showed in a few minutes hardness of the abdominal walls, coarse per-

spiration and ashy appearance, a weak and rapid pulse. The pain, and later also the vomiting were stopped by a hyperdermic of morphine; within three hours the temperature began to rise, hiccough set in at 5 P.M.; the patient became comatose, the radial pulse was lost, Cheyne-Stokes respiration appeared, and at 5.30 he died.

The autopsy was made two hours *post mortem*. There was a perforation of the sigmoid flexure, 10 inches from the anus, about 4 cm. long, with everted deeply injected mucosa, escape of fecal matter and beginning peritonitic reaction. No evidence of ulceration.

The heart (370 grms) showed considerable fat; muscle and fat of the right ventricle being 12 mm. thick. The beginning of the aorta with considerable atheromatous patches. The lungs were retracted and normal.

The kidneys weighed 115 and 105 grms.; the right one had a small cyst and numerous whitish depressions. Spleen 160 grms. with soft pulp and marked trabeculæ.

Liver, 1425 grms., with rather distinct lobules.

The head is injected with 10 per cent. formalin solution. The pia was found slightly edematous, considerably whitened over the frontal regions, the basilar arteries thin; cysterna clear.

The brain weighed 1,380 grms. The frontal lobes separated with slight tearing of the cortex. The spinal cord showed translucency of the pyramidal area from the lower thoracic region downward.

Microscopic examination:

There is a myelitic destruction in the fourth thoracic segment, involving the whole cross-section with the exception of the dorsal two-thirds of the posterior columns, and a few fibers of either pyramidal tract. The secondary degenerations are (See the figures of C_2, C_8, Th_4 and L_4):

(a) Below the lesion:

1. Most of the fibers of the crossed pyramidal tract.

2. The fibers of the direct pyramidal tract, soon disappearing among the antero-lateral ground-bundles, latest in the medio-ventral angle.

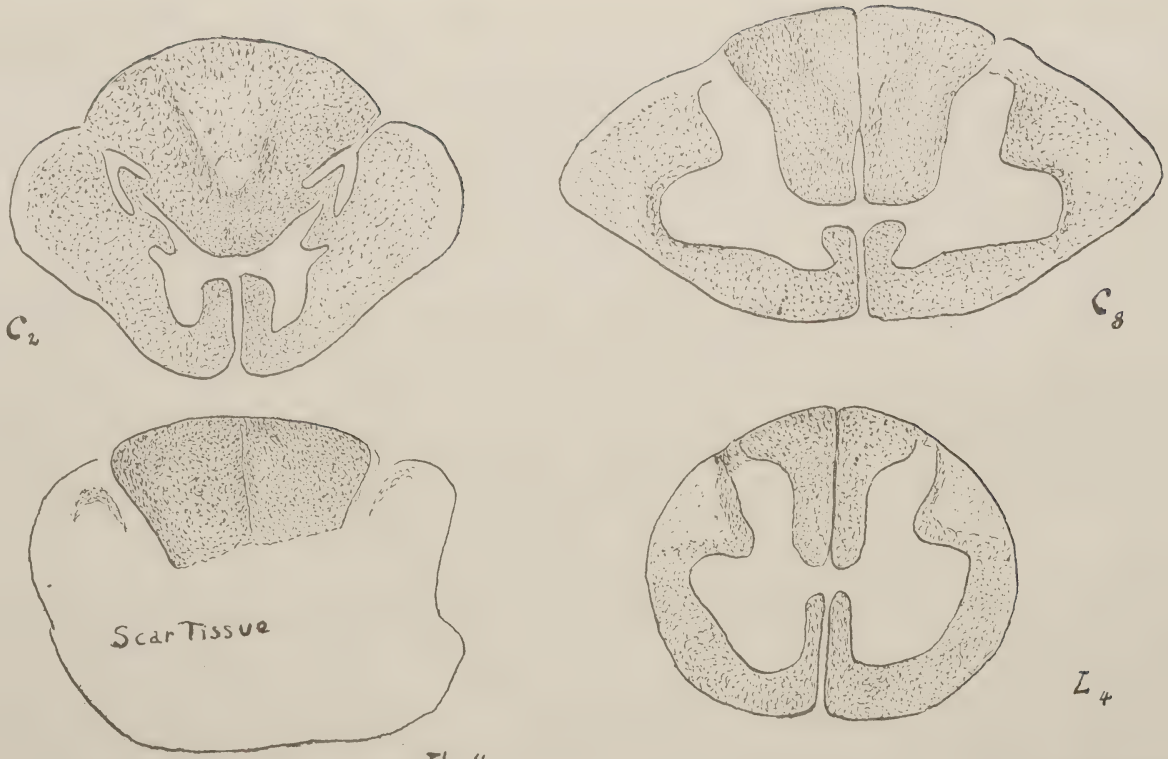
3. The short paths, for several segments in a figure resembling that of Gowers' field.

(b) Above the lesion:

1. The direct cerebellar tract and Gowers' tract are easily followed into the medulla oblongata, but lost from the auditory segment, even in the velum anterius.

2. In the posterior columns a Y-shaped field at the middle of the median septum (in the upper cervical segments).

The great difficulty of final anatomical, physiological and



Sections from the second and eighth cervical, fourth thoracic and fourth lumbar segments, showing the extent of the lesion and the secondary degenerations.

To Journal of Nervous and Mental Disease

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psychological correlation in matters of the spinal cord, pointed out in my "Review of the Data of Modern Neurology"[2], cannot be relieved by cases of this kind, but they assure points for focal diagnosis of some value.

There is, however, a group of cases which Van Gehuchten has collected and discussed[3] with reference to the isolated affection of pain and temperature sense in compression and traumatism of the spinal cord. Van Gehuchten properly distinguishes the cases of Bruns, Vines, Edsall and Marinesco and his own as dissociations covering the whole area below the point of lesion, from the cases of Minor, and Kahler and Pick, which are most likely to be attributed to a localized hematomyelia, and refer only to a small area above the lesion. There are further the cases of J. H. Lloyd[4], which show a one-sided dissociation in the unilateral traumatic lesion of the cervical cord; and, further, a very interesting case by Henry Hun[5] in which the unilateral dissociation was found after a lesion in the medulla oblongata; and a similar case of Wallenberg[6].

All these cases are in favor of Van Gehuchten's view that the conduction of temperature and pain sensations depends on a path in the lateral columns, though probably only in the region of that of Gowers. They also show that Edinger's view of the existence of a decussating sensory path in the cord requires modification and at least restriction to pain and temperature sense. Beyond this, the question remains in suspense as long as the Brown-Séquard symptom-complex is not elucidated.

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[2]Journ. of Comparative Neurology, Vol. viii., p. 299.

[3]"La dissociation syringomyelique de la sensibilité dans les compressions et les traumatismes de la moelle épiniere et son explication physiologique," par A. Van Gehuchten. Extrait de la Semaine Médicale, 1899.

[4]Dr. J. H. Lloyd kindly drew my attention to his two publications: "A Study of the Lesions in a Case of Trauma of the Cervical Region of the Spinal Cord Simulating Syringomyelia" (Brain, 1898, p. 36 and 37), and "A Study of the Lesions in a Second Case of Trauma of the Cervical Region of the Spinal Cord, Simulating Syringomyelia. Jour. of Nerv. and Mental Dis., Vol. xxvii, 1900, p. 65-73.

[5]"Analgesia, Thermic Anesthesia and Ataxia, resulting from Foci of Softening in the Medulla Oblongata and Cerebellum due to Occlusion of the Left Inferior Posterior Cerebellar Artery. A Study of the Course of the Sensory and Co-ordinating Tracts in the Medulla Oblongata." N. Y. Med J., April 17, May 1 and 8, 1897.

[6]"Anatomischer Befund in einem als 'acute Bulbäraffection (Embolie der Art. cerebellar post. inf. sinister.?)' beschriebenen Falle." Arch. f. Psych. vol. 34, p. 923-959.

A CASE OF COMBINED SCLEROSIS OF THE SPINAL CORD.

By Frederick T. Simpson, M.D.,

visiting physician to the hartford hospital.

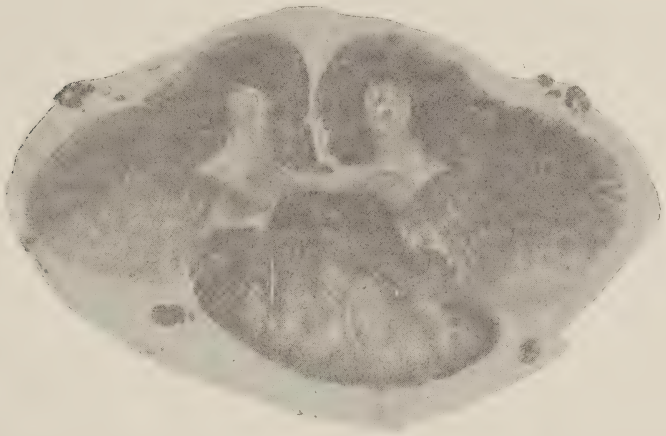
G.L.C., female, of native New England stock; age at death fifty-seven; married twenty-five years, but for fifteen years or more separated from husband on account of his intemperance; mother of two healthy children, twenty to twenty-three years old; no history of miscarriages; no history of syphilis obtainable. For fifteen years, or more, the mother had supported the children by sewing in certain well known families in Hartford among whom she was much esteemed on account of her skill, her industry and her vivacity.

About the age of fifty, she began to notice weakness in her feet and numbness, together with some unsteadiness in walking. These symptoms at first slight, slowly increased, and with them her general health deteriorated. She lost flesh and strength, and became nervous and irritable. She kept on working, however, in one way or another for three or four years. I saw her first in March, 1899, after the disease had been five years in progress.

Status praesens. A woman of rather below medium height, of slender build, dark complexion, of the nervous temperament. Marked pallor is noticeable, and general emaciation. Mind clear, and no marked loss of mental faculty, except control of feelings. Is impatient and irritable and gives way to grief or anger frequently. Sleep normal, sometimes headache and backache occur. Pupils react sluggishly—no myosis nor mydriasis. Grip alike on both sides 35, or about normal; considers her hands all right. No anesthesia nor paresthesia of upper limbs, slight tremor of fingers. Knee-jerks exaggerated, ankle-clonus present, marked ataxia and weakness of legs, but can walk alone. Complains of much numbness in feet and loss of control, but no impairment of perception of touch, pain, or temperature changes to be detected anywhere. Bowels badly constipated and a feeling of "no power" in them. Appetite very poor and digestion bad. No disease of heart, lungs, or kidneys to be detected. Blood examination made by Dr. P. D. Bunce showed normal relations of red and white cells to exist.

During the following six months her condition gradually grew worse, but she still walked with assistance and rode out. In September, she had a sudden attack of complete motor and sensory aphasia with deviation of tongue to right, and dilatation of right pupil. No paralysis of arm or leg. In two or

three days, could talk jargon and could understand and obey a very short spoken command, but not a written one. For two or three weeks she suffered with word deafness and paraphasia, and was given to outbursts of anger and grief manifested by shrieking and crying, and due evidently to the fact that she couldn't understand, or be understood. This condition steadily improved until she was able to speak and hear as well as before. She never had any delusions, hallucinations, expansive ideas, nor any form of melancholia. Her outbursts of anger and grief, and a certain impatience, restlessness and desire for change of conditions were manifestations such as might be expected in a spirited woman of nervous temperament undergoing such an experience. The condition of wasting and weakness



Section from upper thoracic cord showing greatest extent of lesions.[1]

of legs went steadily on until she stopped walking and only moved from bed to chair or lounge. I lost sight of her now for nearly a year. In February, 1901, I was summoned again and found her bedridden, with complete paraplegia and paralysis of sphincters. An immense bedsore existed over the sacrum. Sensory disturbance of legs not marked. The arms were not affected. The mind was clear. Emaciation was extreme. Death from exhaustion occurred within two weeks.

Permission could be obtained to remove only the spinal cord, which was cut off in the cervical region, and sent to Dr. J. R. Hunt, of New York, whose report is substantially as follows:

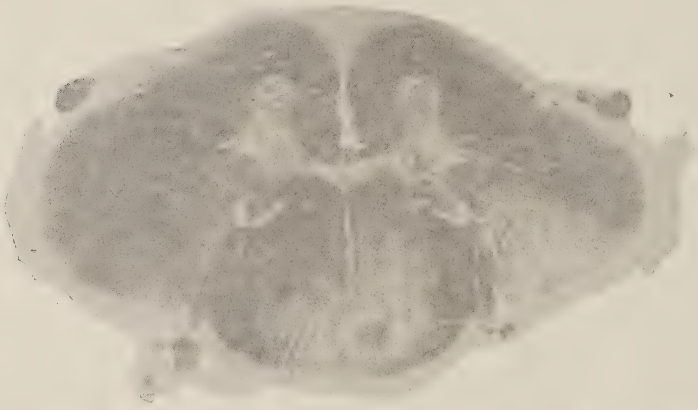
"Cord received in formalin being severed at about the

[1]Photographs by Dr. B. H. Buxton, New York.

seventh cervical segment. The cord throughout to the naked eye appeared very small.

"Findings, Nissl: Throughout, at all levels examined there were noted acute changes in the cells of the cord, particularly in the anterior horns, consisting of chromatolysis, occasionally perinuclear, sometimes peripheral, sometimes irregular shown by a dusty appearance of cell protoplasm in contrast to the sharply defined Nissl granules. A swelling and irregular contour of the nucleolus and an occasional peripheral or excentric position of the nucleus. The above considered to be acute, and produced by conditions occurring not long before death, as decubitus, cystitis, etc., and having no connection with the affection of the cord.

"In addition the cells of the anterior cornua contained ex-



Section from upper thoracic cord showing greatest extent of lesions.

cessive pigment (pigmentary degeneration) so common in advanced life, and chronic cord affections.

"Marchi sections are marked by almost entire absence of acute degeneration. Here and there in the degenerated areas, compound granule cells are found (intensely black) and a few degenerated nerve fibers.

"Weigert and Pal sections show very well the degeneration which corresponds to the so-called quasi-systemic form of Putnam and Taylor, and which is found in the crossed pyramidal tracts on both sides, much more marked in one than in the other, and in the posterior columns along the posterior median fissure.

"The degeneration in the posterior columns begins well above

the lumbar enlargement, thus explaining the exaggerated reflexes. Another histological peculiarity is the vacuolation of the degenerated areas. This is almost pathognomonic of this condition and remains to be explained, and is in marked contrast to tabes, lateral sclerosis, and other forms of systemic cord disease.

"The blood vessels show thickening especially in the gray matter in the ramifications of the anterior commissural arteries, but nowhere could we demonstrate signs of arterial occlusion (endarteritis obliterans).

"There is but slight tendency to neuroglia thickening in the degenerated areas. This is peculiar and tends to produce the above vacuoles. The degeneration of the posterior columns not only bgins high up, but increases as we ascend the cord."

Dr. C. L. Dana, to whom I gave the history of the case, and who examined the specimens, wrote: "It seems to me it is a case of combined sclerosis of the chronic type—not of the type of subacute combined sclerosis, such as I have reported a good many cases of, and such as is usually associated with anemic and cachectic states. The clinical diagnosis of your case, I should judge, was one of chronic ataxic paraplegia. These cases in my experience all belong to either modifications of locomotor ataxia, forms of dorsal myelitis, of Friedreich's ataxia, or of general paresis with spinal complications. It is asserted there is another type, however, not belonging to any of these. I do not think the picture of it clinically has yet been made out, and your case, if it is not one of the group I have mentioned, may perhaps help to clear up this matter."

To the writer, the foregoing case appears to stand midway between the two groups of chronic and subacute combined sclerosis, having features of both, and therefore contributing support to the view which would include the former in the latter. Age and individual resistance appear to be powerful factors in modifying the course of the disease.

NEW YORK NEUROLOGICAL SOCIETY.

October 7, 1902.

The President, Dr. Joseph Collins, in the chair.

A Case of Centralized Scleroderma.—Dr. B. Sachs presented a lady, twenty-four years of age, whom he had first seen six years ago with ordinary hypochondriacal neurasthenia. When next seen, last year, she stated that during the past few years she had noticed that the upper and lower extremities had begun to be stiff and more or less painful on movement. She was not aware at the time that there was anything especially wrong with her face. At present the face shows a very marked form of scleroderma and she also has distinct sclerodactyly. Six months ago there was so much retraction of the upper lip as a consequence of the retraction of the skin, that the gums were constantly exposed. The hands showed tenseness and glossiness of the skin, attenuation and clubbing of the fingers, and an apparent subluxation of the middle finger at the metacarpo-phalangeal articulation. An X-ray photograph shows that the latter is due to the wearing away of the bone under abnormal pressure. There is also a general scleroderma in this patient extending from the forehead to the middle of the abdomen. The lower extremities are only sclerodermatous in certain areas. There are also some areas of leukoderma. The speaker said that this woman had shown a certain amount of improvement under thyroid medication. She had taken as much as 18 grains a day without detriment, and had also had warm baths and exercises with the object of improving the condition of the integument and underlying tissues.

Dr. George W. Jacoby said that he had been one of the first to act upon Dr. Sachs' suggestion regarding the use of the thyroid extract, and he was convinced that this treatment accomplished something. About a year ago he had himself reported two cases, in children, in which the skin had become almost perfectly pliable as a result of thyroid treatment. The changes in the fingers had been very much more marked than in the case now under discussion. Of course, these pathological conditions did not retrograde. He did not think the pressure of the retracting skin was sufficient to explain the marked bony changes observed. In his opinion, the thyroid treatment was the only one that held out any prospect of success, and it was particularly useful in children.

Dr. Joseph Fraenkel said he believed there were two types of scleroderma, the localized and the generalized. The latter appeared to him to be an expression of a rheumatic tendency. For the last four months he had had a case under observation which had done very well under anti-rheumatic medication, particularly the use of the salicylates. Ordinarily the thyroid treatment seemed to be the best method.

Dr. Joseph Collins said that he had had some experience with the thyroid treatment, and while he had observed improvement the results were not at all comparable with those reported by Dr. Sachs and Dr. Jacoby. All that he thought the thyroid did was to diminish the subcutaneous fat. This, of course, made the skin much more pliable over the affected area, and reduced the mask-like appearance of the face. In his opinion scleroderma was a disease of the spinal cord and of the sympathetic fibers and cells within the spinal cord. The symmetry, chronicity, course and termination were all explicable on this theory.

He hoped soon to have an opportunity of making a post-mortem examination upon a marked and advanced case of general scleroderma. In that patient sensory changes were occurring which pointed strongly to involvement of the conducting pathways of the spinal cord. As young persons have a marked accumulation of subcutaneous fat, and this fat could be very readily increased or diminished, it was easy to explain the good results just reported by Dr. Jacoby.

Dr. Sachs said that in the patient he had just presented there had been an extremely disagreeable appearance of the face a few months ago resulting from the attenuated state of the nose, a part having very little subcutaneous fat; yet this part had very decidedly improved under the thyroid medication. He would be greatly surprised if scleroderma proved to be an affection of either the spinal cord or of the sympathetic system. The disease seemed to be diametrically opposite to two other diseases—acromegaly and myxedema. He was inclined to think that scleroderma was possibly a general glandular affection involving not only the skin but the subcutaneous tissues, and even the bones.

Myotonia or Hysteria.—Dr. Edward D. Fisher presented a young man who had first come to his clinic about one week ago. The man was twenty-one years of age, an electrician by occupation. He was apparently well up to the time of enlistment in the Spanish-American war. Over one year ago the left foot began to twitch on attempting to walk. In February, 1902, the right leg became involved, and later the muscles higher up, even in the back. At present all of the muscles below the ribs become more or less contracted on attention. There is no loss of sexual power or loss of sensation. The electrical reactions are normal. On standing up there is a twisting of the body and spasm of the feet. The process had lately extended upward, so that there was now a mild affection of the muscles of the hands and arms. The diagnosis seemed to rest between myotonia and some functional disorder.

Dr. B. Sachs said he had had an opportunity of examining this man about two weeks ago, and had been impressed with the possibility of its being a hysterical condition. He had examined the muscles electrically, and had found them abnormal. There were distinct evidences of a myotonic reaction, a long-continued wave which started in one part of the muscles and spread up slowly between the electrodes. Moreover, with the object of excluding hysteria he had at first applied a current which was very mild, not letting the patient know that he was doing so. As the current was gradually increased up to the point which should produce a muscular contraction, this wave-like contraction was observed.

Dr. J. Fraenkel remarked that he had seen this patient at the clinic, and that Dr. J. Ramsey Hunt had been unable to obtain the myotonic reaction.

Dr. George W. Jacoby said that these cases emphasized the difficulty of making the differential diagnosis between myotonia and hysteria. He had presented a case of myotonia acquisita to the American Neurological Association. All of the myotonic symptoms were present, and there were the electrical and mechanical reactions characteristic of myotonia. A microscopical examination, however, yielded results that were difficult to reconcile with this diagnosis. Dr. Jacoby said he made a bad prognosis, and had subsequently learned that the man after winning a law suit against a railroad company became perfectly well.

Dr. Collins thought if the diagnosis of hysteria major was to be made there should be present more stigmata than merely myotonia.

Dr. E. D. Fisher said that the general aspect of the patient had led to the suspicion of a neurosis, but after careful examination the

diagnosis of myotonia had been made. The electrical examination had been made last June, and at that time no electrical changes were observed. The man's appearance was certainly that of a neurotic individual.

Radiographs of a Tumor of the Brain.—Dr. George W. Jacoby presented some X-ray pictures from a recent interesting case. He said that he had been taking these pictures in such cases for a number of years, but until now the results had been very disappointing. The diagnosis in the present case was a tumor of the brain in the mid-Rolandic region, and the patient had been operated upon about two hours previously. The tumor had been found in the position diagnosed and corresponding to that indicated by the radiograph.

A Case of Brain Tumor (?)—Dr. William M. Leszynsky presented a man, thirty-one years of age, first seen a few weeks ago. Ten years ago he began to have attacks of headache followed by vomiting. At first, there was only one attack each month, but more recently they had recurred about once a week. Six months ago the headache became more intense, and was associated with vomiting and vertigo, and he was in bed for three weeks. On getting up he had diplopia, and three months ago he became blind, and since then had been unable to walk. There was now moderate general headache and vertigo. There was no history or evidence of syphilis, and no history of alcoholism or of infectious disease. Examination showed no tenderness on percussion over the skull. There was some rigidity of the muscles of the back of the neck; both pupils were dilated and immovable, not reacting to light or to convergence. Reflex winking was absent on the left, but well marked on the other side. There was paralysis of both right and left abducens nerves, paresis of both internal recti and an inability to converge. Vision was completely absent in both eyes and there was marked neuro-retinitis on both sides, bnt no choked disk. There was partial paralysis of the seventh nerve and of the orbicularis palpebrarum. There was an actual deviation of the tongue to the left. The grasp was good on both sides. There was no paralysis of the lower extremities, yet he was absolutely unable to stand. There was no apparent weakness of the trunk muscles. The case was presented for diagnosis. The question arose as to whether there were a tumor of the cerebellum originating in the vermis, or one that had extended to this part from the corpora quadrigemina. If the pyramidal tract were not affected it was possible that there might be a secondary meningitis.

Dr. B. Onuf suggested that there might be a tumor of the pons.

A Case of Tabes with marked Bulbar Symptoms.—Dr. I. Abrahamson presented a man of forty-four years, seen at the clinic in the latter part of September. There was a history of marked alcoholism. The present illness dated back to last spring, when diplopia developed, quickly followed by ptosis and lachrymation. The man had lost thirty pounds since that time, and difficulty in mastication and in urination had developed, along with hoarseness and various paresthesias of the throat. The temporal arteries were tortuous, the patient was badly nourished and presented evidence of degeneracy. The Romberg symptom was present, and the motions of the eyes were restricted. There was marked wasting of both temporal and masseter muscles, with greatly diminished reaction to both electrical currents. The optic nerve showed beginning white atrophy. The knee-jerks and Achilles reflex were absent on both sides, while the bulbar reflexes were lively. Tactile sensibility was nearly normal. The chief feature of the case was the extensive cerebral and

nuclear involvement. The case was evidently one of tabes with very marked bulbar symptoms.

Differential Diagnosis of Multiple Sclerosis.—Dr. B. Onuf presented a paper on this subject. He quoted from the literature to show that it was probable that this term, multiple sclerosis, referred to a symptom-complex representing most varied pathological processes. The diseases most apt to be confounded with it were diffuse sclerosis, pseudo-sclerosis, cerebro-spinal syphilis, general progressive paresis and a number of other diseases, such as tabes, ataxic paraplegia and transverse myelitis. Diffuse and pseudo-sclerosis had a symptomatology so similar that he did not feel the differential diagnosis could be clearly made. The multiplicity of the lesions of cerebro-spinal syphilis and the recurrence of the symptoms after intervals of comparative freedom from them bore a close resemblance to multiple sclerosis. It was evident, therefore, that the diagnosis must be founded on a complete clinical picture rather than on one or two individual symptoms. The value of antisyphilitic treatment was great as a diagnostic aid, but the results were not wholly reliable, particularly when marked improvement was not noted within a period of about two weeks. Spasticity was just as frequently observed in syphilis, and the intentional tremor he had seen very typically developed in a case of undoubted cerebral syphilis. Fairly developed nystagmus pointed very strongly to cerebral syphilis as against disseminated sclerosis. Scanning speech was a strong symptom in favor of disseminated sclerosis as against syphilis. There were two symptoms which he considered of great importance: (1) the facial expression, and (2) an emotional state associated often with marked euphoria. These two symptoms were relatively rare in syphilis. Optic neuritis was present in a large percentage of cases of multiple sclerosis, and was often the only symptom in the early stages; however, optic neuritis was very common in cerebral syphilis, though it was more apt to be attended by retinal hemorrhages. The fact that multiple sclerosis was often ushered in by some acute disease was a point of some diagnostic importance. The speech disturbance in general progressive paresis was often quite marked, indeed this affection should not often be confounded with multiple sclerosis. The number of spinal diseases that might be simulated by multiple sclerosis was very great. In doubtful cases the presence, besides the spinal manifestations, of symptoms pointing to multiple cerebral involvement would speak in favor of disseminated sclerosis, as would also the symmetry and regular distribution of the spinal symptoms. The speech of bulbar palsy might be very much like that of multiple sclerosis, and the patient might also be distinctly emotional.

Dr. Fisher thought multiple sclerosis was most likely to be confounded with cerebral syphilis and general paresis, and yet it was only at certain stages that even here any difficulty existed. The tendency to stupor and attacks of prolonged sleep, and the ocular palsies were characteristic and were not observed in insular disease. In general paresis the flattening of the face, the tremor and the articulation were quite similar, but the mental state was quite different. At times multiple sclerosis might be confounded with hysteria. The disease being very slow in its progress years might elapse before the true diagnosis of insular sclerosis could be made. It should be borne in mind that insular sclerosis sometimes occurred in young people, at the age of sixteen or eighteen years, a point of differentiation from cerebral syphilis.

Dr. Onuf agreed with Dr. Fisher that the tendency to somnolence was ordinarily a strong symptom in favor of syphilis, and admitted that it was sometimes difficult to distinguish the disease from hysteria.

Arterial Disease in Comparatively Early Life.—Dr. E. D. Fisher read a paper with this title. He said that if one had not established himself in some definite line of work by the age of forty, he would rarely succeed. This was the law of life. Having excluded syphilis, kidney disease, diabetes, alcoholism and old age there would still be a number of cases of arterial disease having a different etiology. He was of the opinion that cerebral hemiplegia was more than ordinarily common at the present period of our national development. There was an intensity in the pursuit of an object in the Anglo-Saxon race not present in the Latin races; this led us to a very large consumption of tissue. He believed that the so-called "strenuous life" led to a fatty degeneration of the cardiac and arterial muscular tissue. If this view were correct, then the means of prevention were obvious and important. Our social system in the large cities was one of anxiety and overwork. There should be less straining after living, as is the case with the very rich, and a stronger desire for culture and moderation.

Dr. W. B. Noyes raised the question as to whether, in the treatment of so many diseases of the nervous system with strychnine physicians were not committing a grave error. The effect of this drug on the arteries was not closely studied, and it had occurred to him that in cases like those described in the paper, in which there was, in his opinion, arterial disease, the persistent use of a drug like strychnine, which increases the arterial pressure, was actually harmful and more than counterbalanced the beneficial action of the remedy upon the peripheral nerves. He believed that many of those present could bear him out in the statement that iodide of potassium frequently benefited many cases in which there was no syphilitic taint, probably by its effect on the vascular system. He would like to hear from others regarding this view that strychnine and iodide of potassium are, to a certain extent, antagonistic in their action on the vessels.

Dr. Leszynsky said he could not agree with Dr. Fisher in the contention that the so-called strenuous life produces cerebral and arterial degeneration unless this were accompanied by alcoholism, syphilis or some other toxic cause. He had never seen a patient under forty years of age with a cerebral hemorrhage or endarteritis unless there were some discoverable cause which would lead one to the belief that such conditions had previously existed.

Dr. Sachs said it was interesting to consider whether there were really arterial disease in early life sufficient to lead to apoplectic attacks. Personally, he could not recall a single case of cerebral hemiplegia which was not due to arteriosclerosis, syphilis or embolism, or was the accompaniment of renal disease. There was only one other vascular degeneration occurring early in life, *i.e.*, a fatty degeneration of the artery, and which explained the very early apoplectic attacks in children. When he had been able to exclude embolism, renal disease and syphilis he had always come to the conclusion that the case was one of early arteriosclerosis.

Dr. Joseph Collins said that he found it very difficult to talk upon arterial *disease*—not sclerosis. He was thoroughly convinced that chronic degeneration of the tunica medica, arteriosclerosis, was a disease of the strenuous life, and that alcoholism, rheumatism, syphilis and the so-called metabolic diseases had very little to do with this. A deficient heredity was one of the contributing causes. Another was chronic indigestion of any kind, and a third was worry with work. These were more potent causes of arterial degeneration than syphilis, alcoholism and bad habits. The last named conditions caused periarteritis and endar-

teritis. A second great class of cases was dependent upon infections; some would put these as the first and more important class. We had been taught as students that arteriosclerosis was a disease occurring in those past fifty, but he maintained that this was not true, and that when the disease began at that time it was nothing more than a natural process at that age. A person of sixty-five or seventy years of age becoming ill with an infection like pneumonia usually had the disease in an exceedingly mild form. He would further contend that arteriosclerosis was, at the present day, the scourge of humanity, and that there was no organic disease of the nervous system that could compare with it in its effect upon the production of disease.

Dr. Leszynsky thought the last speaker had begged the question with regard to young subjects. If the strenuous life reacted upon digestion and interfered with assimilation it caused an intoxication or an infection, and in this way set up an arteriosclerosis. He did not think any proof could be adduced to show that the strenuous life alone produced arteriosclerosis.

Dr. Fisher, in closing, said that a man over forty years of age would rarely start out in a new line of work, although he might continue to do much very good work in old channels up to quite late in life. If he had understood the remarks of Dr. Collins, he did not think their opinions were very much at variance on this topic. By arterial disease he meant any morbid condition of any part of the arteries.

CHICAGO NEUROLOGICAL SOCIETY.

Joint Meeting with the Chicago Medical Society.

April 2, 1902.

The President, Dr. Daniel R. Brower, in the chair.

Definition and Pathology of Neuritis.—Dr. Archibald Church spoke on this phase of the subject. He said that some fifteen years ago nothing was heard of neuritis. There was even a general feeling that the nerves were to some extent immune against the processes of inflammation. Ten years ago a definition of the term neuritis would have been of the greatest ease; if one had said that it meant inflammation of the nerve, he would have covered the ground. This condition, at first apparently simple, had been found to be complex, and now the term neuritis was a generic one. It had to some extent been stretched out of its proper significance.

He divided cases of neuritis into the true form, marked by inflammation in the nerve or its fibrous support, and those forms of neuritis characterized by degenerative processes. True neuritis, the first variety, was generally confined to a single or to several nerves, but it did not, as a rule, involve a great number of nerves. It was inflammatory, and usually arose from the surrounding tissues involving the nerve by extension, by continuity or contiguity.

It may be secondary to a new growth. Any variety of neoplasm in the neighborhood of the nerve may by infiltration set up irritation and terminate in actual inflammation of the nerve. In some instances the nerve was invaded by the tubercle bacillus; in others by the germ of leprosy. Syphilis involved the nerves much more frequently than was supposed. Sometimes when sciatica does not yield to ordinary methods of treatment, a course of mercury is proper. In leucocythemia and a number of other disorders of the blood characterized by dyscrasia and disintegration of blood elements, portions of the peripheral nerve were inflamed, presenting a true neuritis.

Attention was directed at considerable length to the degenerative form of neuritis. The speaker said it was more common than the inflammatory variety. The degenerative forms of neuritis were generally the result of toxic causes or elements, the principal ones of which were lead and alcohol. Various forms of cachexia, especially syphilis, were attended by manifestations of degeneration in the motor portions of the peripheral apparatus.

The character of the degeneration in multiple neuritis was practically a Wallerian degeneration. Multiple neuritis must be looked upon as the result of systemic poisoning, and as a systemic disorder involving all portions of the nervous apparatus. The chief toxic substances which give rise to multiple neuritis were alcohol, lead, arsenic. Reference was made to the occurrence of the epidemic in Manchester, England, in connection with beer-drinking.

Owing to the idiosyncrasy of a patient, mercury given in the usual manner had been known to set up all the symptoms of multiple neuritis, the neuritis subsiding upon the withdrawing of the mercury. Other agents were mentioned as toxic substances capable of setting up multiple neuritis, among them phosphorus, sulphur, *etc.*

Among the autotoxic causes of multiple neuritis, he spoke of diabetes. In chronic interstitial nephritis, owing to a toxic condition, there

was disturbance of the nervous apparatus, showing itself in vague, yet when carefully considered, highly suggestive sensory manifestations of disturbance of the nervous apparatus. Another group of autotoxic factors arose from intestinal conditions. Individuals subject to dysenteries, parasites, and chronic intestinal disorder, might develop multiple neuritis. All of the infections practically were capable of inducing multiple neuritis through their toxic action.

In pernicious anemia one might have all the symptoms of neuritis which were attended in the majority of cases by histological changes in the spinal cord.

In old age, in patients in whom there was extreme arteriosclerosis, there were often manifestations of multiple neuritis, the disease arising from a number of causes. For instance, the artery which supplied the nerve, owing to an atheromatous condition and a sclerotic state, did not deliver enough nutrient material to the nerve, so that nutrition was disturbed locally.

Symptomatology, Diagnosis and Differential Diagnosis of Neuritis.—This was discussed by Dr. Sydney Kuh. He called attention to the fact that in toxic neuritis—and possibly in other forms—changes are found not only in the peripheral nerves but in the cells of the central nervous system as well, and that a large number of nerve fibers may be found degenerated in cases in which *intra vitam* no signs of disease in them could be detected. The disturbances of sensibility—both subjective and objective—were first described, then those of the higher senses, those of motility, of electric irritability, of coördination, of reflexes, of trophic, secretory and vasomotor functions, *etc.* Korsakow's psychosis as a symptom of multiple neuritis was discussed in detail and the constitutional symptoms accompanying neuritis briefly mentioned.

In speaking of the diagnosis of neuritis rules were given for its differentiation from rheumatism, disease of bones and the periosteum, hysteria, neuralgia, cerebral disease—especially that involving the facial and oculomotor nerves,—acute and chronic poliomyelitis, locomotor ataxia, muscular dystrophy and polymyositis.

Treatment of Neuritis, Other Than Surgical.—Dr. Elbert Wing said that in the treatment of neuritis it is of first importance to make a correct diagnosis; to determine that the case is one of neuritis, and whether simple, multiple, toxic, acute or chronic. The objects of treatment are removal of the cause, and restoration to normal conditions. When due to pressure, instrumental or from foreign growths, removal of the cause is often a simple matter, but may be difficult. A similar statement applies to cases due to, and complicated by, gouty or rheumatic states. In these cases disappointment is not apt to follow correct diagnosis and efficient methods of treatment. In cases due to mineral poisons the detection of the source of the offending element may at times be difficult, and in alcoholic cases clever deception on the part of the patient may increase the difficulty. In cases which are really those of the parenchyma—true degeneration, the pathologic conditions of course continue long after the specific cause is removed.

Relief of pain is imperative in all acute cases of severity. For this purpose hot cloths properly applied along the course of the nerve often are of great effectiveness. Counter irritation may answer, but should never be applied in the area of distribution of the affected nerve. In chronic cases the actual cautery is the best counter irritant. In acute simple cases Gowers' liniment is of great value. Sand bags, slings and splints have a useful function. They may promote relief of pain through limitation of motion, and prevent and correct deformities. In

most severe primary cases morphine is needed for the relief of pain. The coal-tar preparations rarely suffice. If used, the doses must be large, frequently repeated, and acetanilid should never be used for this purpose. Morphine, cocaine and other analgesics are most effective when placed, by means of a hypodermic syringe, in the immediate proximity of the affected nerve.

Stretching of the nerve, in simple sciatic cases, by forcible flexion of the trunk upon the extended lower extremities, or of the extended extremity upon the trunk, the body supine, is at times remarkably successful both in relieving pain and promoting recovery. The same is true of efficient massage. The use of electrostatic currents and the Roentgen rays have a useful future in the relief of pain in probably all cases of neuritis. Unfortunately they are not often available for treatment of these cases. In some cases the galvanic current has decided pain relieving power, in others its use increases pain. A safe rule is never to use a current strength which causes a decided increase of pain at the time of its use, or any persistent increase.

The rare forms of neuritis due to syphilis are treated along the general lines of the two diseases and need no elaboration in this connection.

The various forms of multiple neuritis differ radically in their causes, but are so essentially similar in their pathologic conditions in many respects that their treatment is much along the same lines. These lines are removal of cause, prevention of contractures, protection of tender extremities, at times relief of pain, and finally the general tonic treatment of any severe chronic disease in which the essential lesions are degenerative. In this list general tonics take high rank and the salts of strychnia the first. As in any other chronic condition, the form of tonic used must be occasionally varied. In the chronic forms of simple neuritis the use of one grain of blue pill, two or three times daily, and for long periods, produces favorable results. In polyneuritic cases, massage, skilfully used, the proper use of splints and electricity, together with voluntary exercise of muscles, bring about cures even in the worst cases.

Two classes of multiple neuritis require special mention. As a rule, in alcoholic neuritis, alcohol may be withdrawn at once. In the variety of neuritis due to lead, the conventional attempts at specific removal of the mineral are at first demanded.

The causes of death in the fatal cases indicate clearly the special care which is needed, cardiac and pulmonary weakness being induced both directly by the special cause in each case and by the auto-intoxication which may arise in any case. These principles of treatment must be constantly in mind in every case of multiple neuritis. They are usually simple and well understood, but must be the objects of constant watchfulness.

Surgical Treatment of Neuritis.—Dr. Weller Van Hook discussed this phase of the subject, saying that surgery is in a position to be of service in the treatment of the consequences of neuritis rather than in the management of the actual disease itself, whether acute or chronic. In the acute form of the disease, aside from those instances in which a suppurative lesion is present, very little can be done by surgical means for the relief of the patient, except to afford some aid by rest. Where pressure upon a nerve is superinducing neuritis, or neuralgia, the case belongs within the province of surgery. Where fractures primarily or secondarily involve large nerve trunks, operative procedures are frequently needed; for example, where a fractured clavicle

presses upon the brachial plexus, relief by elevating the fragments and separating the connective tissue from the nerve is of absolute necessity. Not infrequently the long bones, when fractured, similarly press upon important nerve trunks.

In open injuries, where infection is likely to occur, the utmost care must be taken by the surgeon to prevent infection of the wound, particularly where large nerve trunks are likely to be involved. There may be not only inflammation of connective tissue around the nerve involving the nerve trunk in adhesion, but it is also possible to have from such a wound a migrating neuritis successively involving different nerves. This form of neuritis is supported as a nosological entity by Krehl, who maintains that well-marked cases of the disease have frequently come under his observation.

In the chronic forms of neuritis surgery is of more frequent avail. It is in the treatment of the consequences of peripheral neuritis that surgery finds the greatest scope. In compression of nerve trunks by periostitis, it is admissible to remove the connective tissue around the nerve, as has been frequently done, an operation which is termed neurolysis.

Tumors of a non-malignant character that press upon large nerve trunks, producing more or less of an inflammatory condition, should be attacked by excision, while those which are of malignant character demand removal of the nerve or of a part of the nerve itself.

The method which one should pursue in the treatment of hemorrhages within and about nerve trunks is yet open to difference of opinion. No definite rules have been laid down for the management of cases of this kind.

In the case of chronic inflammation producing neuralgia, a variety of surgical procedures are at the disposal of the surgeon. The stretching of chronically inflamed nerves is no longer practised so freely as was formerly the case; nevertheless, in cases of true neuralgia of the sciatic nerve, Hoffa recommends in selected cases the stretching of the nerve, at first by the subcutaneous method, and later, if necessary, by an open procedure.

The method of extraction of the nerve, exeresis, at first practised by Thiersch, is still in very common use. Undoubtedly, the majority of cases of neuralgia of the trigeminus can be cured by exeresis, and it is by no means necessary that the Gasserian ganglion should be excised in ordinary cases of this disease. On the contrary, this formidable and dangerous operation should be reserved for those cases in which peripheral operations have been thoroughly tried.

When paralysis is a consequence of neuritis, surgery offers relief in many cases by transplantation of nerves or of tendons. Results obtained by this modern method of treatment are very gratifying.

The French school of surgeons, at the head of which is Chipault, have of late shown much enthusiasm in the management of many cases of peripheral nerve disease, particularly of the trophic varieties, by stretching. It is claimed by Chipault that *mal perforant* of the foot can be favorably influenced in many instances by the stretching of the nerves of the leg.

It is particularly in cases where the sciatic has been injured, or where it has been involved in inflammations of the thigh, that nerve stretching has seemed to be of service in *mal perforant*.

Sciatica was discussed by Dr. L. Harrison Mettler. Sciatica in some recent textbooks is still classified under several heads, as for instance primary and secondary. The primary is divided into the idiopathic and

the special forms of neuritis. He thought this was a mistake. He could not conceive of a secondary sciatica in the sense of a mere pain of the nerve caused by some extraneural pressure. If there is disease of the bone, tumor, or other condition causing secondary disease, either of an inflammatory or degenerative type, in the sciatic nerve, it might seem to be a secondary sciatica, but it is only secondary etiologically. It is really a sciatic neuritis; hence he thought so-called secondary neuralgia, or sciatica, as a special class should be dropped from the books. Almost all the cases he had seen of sciatica exhibited more or less the symptoms of sciatic neuritis. The symptoms which usually accompany so-called ordinary idiopathic neuralgia of the sciatic nerve were those that are characteristic of sciatic neuritis. These were mentioned.

As to the treatment in cases of sciatic neuritis, where there was a rheumatic diathesis, he obtained the most favorable results from the use of the salicylates, pushing them until he got a decided physiological effect. He had not seen such favorable results from the use of mercury or anti-syphilitic treatment. Sciatica was not usually caused by syphilis.

Neuritis of the Fifth Nerve.—Dr. Daniel R. Brower detailed the report of a case of this form of neuritis. In taking the patient's history he inquired very carefully into his antecedents, particularly as to syphilis. The patient at first denied syphilis. In early life the patient had had two or three attacks of rheumatism. He was quite anemic. Dr. Brower did not know just how to begin treatment of the case, but first treated him on the anemic theory, and tried to build him up by the administration of appropriate hematinics; at the same time using galvanism. Under this treatment the patient grew worse. He was then treated on the rheumatic line, salicylates and iodides being given. The patient did not improve under this treatment, but became worse. Finally, believing that the patient was mistaken as regards syphilis, he gave him the mixed treatment for syphilis, and the pain disappeared as if by magic. Again he asked the patient in regard to syphilis as the probable etiological factor, and he admitted it.

He had had within three months two cases of neuralgia or neuritis of the fifth nerve, both syphilitic. It was rare in his experience for this nerve to be attacked by the syphilitic poison, but these cases coming so close together were interesting as well as instructive.

Dr. C. P. Pruyn said that dentists had a good deal to do with neuralgia of the fifth nerve. He was a little surprised on being told by a prominent neurologist that neuralgia of the fifth nerve was seldom caused by tooth irritation. Frequently the patients consulted dentists for relief after they had gone through the gamut of the general treatment by physicians, and tooth irritation was found to be the cause. Often there was calcification of the tooth pulp, sometimes complete, at other times simply a calcified nodule, which caused the whole disturbance. The removal of this nodule effected a cure.

Dr. O. B. Will, of Peoria, believed that in the acute or primary form of neuritis, where the patient was suffering much from neuralgia, the best treatment was the administration of chloroform or ether. He used this altogether in his own case, and his professional friends had adopted it, with good results. The chloroform should be administered *per orum,* hypodermically, or by frequent inhalations. He thought there was no agent that would do as much in bringing about permanent relief in the class of cases under discussion as this agent. It should not be administered, however, to the point of anesthesia, but the system should be more or less saturated until pain was relieved.

Dr. A. W. Baer had used the salicylates in the rheumatic form of

neuritis, with beneficial results, especially if not pushed to the point of interfering with the functions of the stomach. He had never obtained much benefit or relief in these cases from external application, outside of heat and cold, except from the effects of rubbing oil on the parts affected. If the neuritis were of traumatic origin, the interrupted galvanic current was by all odds the best, and he had obtained some excellent results from it. Sparks from the static current in cases of toxic neuritis had given excellent results.

Dr. Julius Grinker thought motor disturbances were often the most prominent symptoms in cases of neuritis. Diphtheritic paralysis was nothing but a neuritis following diphtheria infection, manifesting itself by motor disturbances principally, the sensory disturbances taking a back ground, or perhaps not being noticed. Faucial paralysis follows diphtheria. He had seen a staggering gait develop in children after diphtheria. This was nothing but a symptom of multiple neuritis. It is well to recognize these forms of neuritis, because by so doing a life could occasionally be saved. Physicians hear of sudden deaths of children after diphtheria. A child is said to get well. walks about, but cannot swallow readily; fluids are regurgitated, and the child suddenly dies. Heart failure is said to be the cause of death. He thought possibly many of these cases were due to vagus involvement, the neuritis having developed and progressed rapidly and involved the vagus, killing the child, the nature of the trouble being unnoticed by the physician. Symptoms should be looked for in cases of diphtheria even after recovery has occurred. He urged that the reflexes be tested, as the first symptom noted after diphtheria of oncoming neuritis was absence of reflexes. When the reflexes are diminished or absent, one should look out for neuritis. It might develop rapidly and kill the patient.

Not long ago he was called to see a case in private practice. A child commenced to stagger two weeks after diphtheria. The mother noticed that the child's eyes seemed to be in the same position; in directing the child's attention to anything, it would stare in front of it and not move a muscle. He was called and recognized paralysis of almost all the ocular muscles, also paralysis of the lower extremities. He considered it a neuritis following diphtheria. He gave the child strychnia in large doses, and it is well today. He does not believe in giving the usual textbook doses of 1-120 of a grain, but the 1-30 of a grain, three times a day.

Dr. G. W. Hall said he had had the privilege in the last few months of seeing at the County Hospital two extreme cases of peripheral neuritis involving all the limbs of the body. The first patient, when admitted to the hospital, was unable to move either a hand or foot, and had no sensory disturbances. In the other case both arms and lower limbs were involved completely, with facial paralysis on one side, finally extending to the other side, with complete motor paralysis of all the limbs. There were no sensory disturbances. It was important to remember in making a diagnosis that sensory disturbances are not necessarily present. He believed that next to alcoholism sensory disturbances were more frequently present in peripheral neuritis than in any other form. He emphasized the great difference as to the presence and absence of sensory disturbances in the different forms of neuritis, saying that in neuritis following lead poisoning the sensory disturbances were very slight as compared with the motor. In neuritis following alcoholism it was very rare that we did not have sensory disturbances present to a greater or less extent. He believed in many cases paralysis comes on almost simultaneously with sensory disturbances.

Dr. Liston H. Montgomery would not care to use such large doses of strychnia as mentioned by Dr. Grinker. A formula which he had used with beneficial results is one composed of menthol crystals, dilute phosphoric acid, and a few drops of tincture of aconite with alcohol.

He thought it was an oversight that no one had mentioned rheumatism as a possible etiological factor of single or multiple neuritis.

Diet was a very important feature in the treatment of many cases of neuritis. Recently he had a patient who had suffered from multiple neuritis which had defied every form of treatment for eight years. One of the first questions he asked was whether any of the former physicians had ever prescribed a diet for the patient, and the patient replied that he was allowed to eat anything he desired. The speaker eliminated a number of articles from the diet, and along with the preparation mentioned he gave iodide of potassium in twelve grain doses, three or four times a day, and under this treatment the patient recovered.

Dr. Grinker did not consider the thirtieth of a grain of strychnia, administered to a child of eight or twelve years of age, a large dose. He had used it in such doses in several cases with excellent results.

Dr. I. A. Abt believed that strychnia is frequently given to children in too large doses. Not long since he saw a child at a hospital suffering from extreme rigidity and from opisthotonos. The symptoms made him think of tetanus, possibly a meningitis. He looked at the history sheet and found that the child, an infant not more than a year old, was getting nearly the twentieth of a grain of strychnia in twenty-four hours, and was suffering from strychnine poisoning. During the infantile period he believed great caution should be exercised in the administration of this drug.

Dr. James W. Walker mentioned the use of the Paquelin cautery in the treatment of neuritis, saying that he had obtained excellent results in the relief of pain. He could say nothing regarding its effect upon the inflammatory process in acute neuritis. It seemed formidable to the average practitioner to see it used. The lightest possible touch was all that was necessary. It could be used once daily, or once every other day, two or three strokes being made in the vicinity of the pain, affected joint or nerve.

Dr. Thomas L. Gilmer said he had been a sufferer from neuritis, especially of the sciatic form. A short time ago Dr. McArthur gave him a prescription for local application which did him more good than any one thing he had used for a number of years. This prescription was: menthol, 8 grams; oil of gaultheria, 30 grams, and creasote, 2 grams.

Dr. Kuh, in closing the discussion, agreed with Dr. Montgomery that rheumatism and a number of other causes might produce neuritis, Bright's disease amongst others.

With reference to the remarks of Dr. Grinker, he said that if his (Grinker's) patient had paresis of the lower extremities, associated with total paralysis of the movements of the eyeballs, the patient probably did not suffer from a diphtheritic neuritis. Total paralysis of all the extrinsic muscles of the eyeballs could hardly occur in neuritis.

Periscope.

Rivista mensile di psichiatria forense, antropologia criminale e scienze affini.

(Vol. V, 1902, No. 1, January.)

1. Tattooing of Neapolitan Criminals. A. DeBlasio.
2. Restricted Responsibility. P. Penta.

1. *Tattooing of Criminals.*—A highly suggestive sketch touching upon tattooing as expressive of the psychology of the criminal; shedding, *en passant,* some interesting light on the well known Camorra, an organized body of Italian criminals. The four individuals who constitute the subject of the study are brought forward as a proof of the author's contention that in many cases tattooing tells the story of the individual, certainly to those who are able to decipher it. Moreover, tattooing very often presents in figures and letters the criminal's aims and intentions as to the future, and may thus assist the authorities in preventing him from carrying the latter into effect. This refers especially to tattooing expressing revenge (vendetta).

2. *Diminished Responsibility.*—According to a great number of prominent alienists, among them Schrenck-Notzing, diminished responsibility is to be charged in cases of even slight mental affections, in incipient stages of others (as that of general paralysis), in simple psychic degeneration without delirium, in cases on the border line of insanity, those of sexual perversion, chronic alcoholism, neurasthenia, general epilepsy, etc. According to Mercier there is a certain sphere of activity in which every insane individual is to be held irresponsible, but outside of which any act of his may be punishable by law; so that but a few insane can be held entirely irresponsible. The author inclines to the idea that the physician should not be permitted as such to decide on the degree of responsibility in these cases, but should simply and clearly describe the patient's mental state, leaving to the judge the application of the law.

(Vol. V, 1902, No. 2, February.)

1. Examination of Four Crania of Insane Criminals. G. Antonius and M. Falciola.
2. Asylums for Criminals and Departments for Insane in Penitentiaries. P. Penta.

1. *Examination of Four Crania.*—This is a minute and accurate examination of four crania from a collection of skulls of insane in the Voghesa Museum, whereby the author intends to prove not only the existence of degenerative stigmata morphologically, but that of a real anthropological type in a homogeneous group of criminals. The latter all committed homicide, and their skulls present such a remarkable similarity that they can easily be mistaken one for another. The author adds the results of this examination to the numerous proofs of the fact that Lombroso's theory as regards the criminal rests on a solid anatomical basis.

2. *Asylums for the Insane.*—The unprecedented crowding of the asylums for the insane of late years is ascribed by the author chiefly to two facts: the terrible increase of insanity as proven statistically, and the better as well as the more diffuse knowledge of the nature of insanity

itself. To the new school of criminology is due the merit of pointing out the relation between insanity and crime. The asylums for criminals should be restricted only to such insane criminals whose pernicious tendencies, through a certain complexity of circumstances, can never be eradicated; so that their liberation will be fraught with danger to the community; the others, such for example in whom insanity develops during their detention, or those with slight forms of imbecility, may be left in penitentiaries, there to be watched and treated. For these latter special departments should be established in the various detention houses (such are already to be found in some jails in Germany), under the care and guidance of physicians more or less skilled in the problem of treating the insane.

(Vol. V, 1902, Nos. 3, 4 and 5, March, April, May.)

1. The Neapolitan Gypsies. A. DE BLASIO.
2. The Criminal Soldier. F. SAPORITO.
3. Tuberculosis and Insanity. A. GRIMALDI and F. SCOTTI.

1. *The Gypsies.*—An interesting article on the comparative anthropology of the gypsy.

2. *The Criminal Soldier.*—This is a series embracing observations and histories of eighty-five subjects, classified in certain anthropological categories.

The whole series presents first of all a variegated collection of hereditary mental defects, including insanity, epilepsy, hysteria, criminality, alcoholism, consanguineous marriages, etc.—a rich pabulum for the future criminal to grow on. And, as a matter of fact, the subjects themselves show in their anamnestic data the full effects of the hereditary influences, what the author calls an "aberrant" organic development; among these data we find most predominating abnormalities of the nervous system; such as "convulsibility" in the early years, nocturnal enuresis extending even into adult life, somnambulism, vertiginous attacks, etc. Their moral nature, the way "they thought, felt and acted," is fully in accord with what could be expected from their heredity and personal anamnesies. Indeed, the personal observations and a detailed classification of their defects, show that the series ran the whole gamut of degeneracy, vice and crime: habitual alcoholism, general dissolution, extreme irascibility, marked precocious criminality, etc. Special attention is drawn to the fact that both the insanity and the delinquency were of a precocious nature. Finally two tables containing the morphological and functional stigmata of the subjects complete the picture of the series. Among the first are found cranio-facial asymmetries of various forms,—acrocephaly, clinocephaly, prognathism, prominent zygomata, precocious calvities, asymmetrical ears, etc.; then, gynecomastia, disproportionate genitalia, and so on; among the latter, various tics, tremors, nystagmus, strabismus, exaggerated reflexes, ankle clonus, dermographia, hyperesthesia cutis and the like.

The subjoined detailed personal observations of individual subjects of the series are of great interest, but do not lend themselves to abstraction.

3. *Tuberculosis and Insanity.*—The frequency of mental decay among phthisical patients had been observed by many investigators. Féré had long ago found phthisis quite prevalent among epileptics, while in tubercular families nervo-psychic degenerations are far from being rare. One of the present authors presented some time ago the genealogical tree of a paranoiac of a greatly tainted family, whose decadence dates from a tuberculous forefather. Tuberculosis may well be placed in the same cat-

egory with syphilis, alcoholism, and pellagra among the pernicious factors in the development of the race. The present investigation is based on statistics supplied by several asylums which were addressed on the question of insanity and phthisis. (A procedure which would be of great value if instituted by the proper authorities here.) Out of a total of some sixteen thousand insane there were over nine hundred phthisical sufferers of whom eight hundred died during the last half decade. Unfortunately no definite data could be obtained as to the period when tuberculosis developed (before or after the appearance of insanity), although it may be stated in a somewhat general way that in the majority of cases phthisis developed while in the institution. It was shown clearly that hereditarily there was in the majority of cases a neuro-psychopathic tendency in the family; the phthisical process exerted generally a very deleterious effect on the course of the mental malady; while some few cases showed some amelioration and even mental lucidity in the last moments of life. In view of the fact that the unfortunate insane in his search for a remedy for mental rehabilitation falls a prey to a far more terrible physical malady, the author recommends that insane asylums should be provided with special isolation wards for phthisics, and even that special asylums be founded for the care of phthisical insane.

In connection with this it is interesting to compare the Report of the Tuberculosis Committee of the Medico-Psychological Association of Great Britain and Ireland, abstracted in the November issue of the JOURNAL. ALEX. ROVINSKY (New York).

Neurologisches Centralblatt.

(1902, Vol. 21, No. 15,, August 1.)

1. An Unusual Case of Facial Spasm (Myokymie) Occurring in the Distribution of the Left Facial Nerve. M. BERNHARDT.
2. On Insanity Following Experimental Autointoxication. Thyroid Psychoses in Dogs. F. BLUM.
3. On the Subcortical Origin of Isolated Muscular Spasms. J. SORGO.

1. *Facial Spasm.*—The presence of persistent hypertonicity of the facial muscles of the left side, associated with constant fibrillary tremors in the same distribution, in a woman of twenty-eight years, disappearing after two months, led the author to class this under those already reported as myokymie. The distribution affecting the face is unique.

2. *Experimental Psychoses in Dogs.*—Blum, after an extensive series of experiments on dogs comes to the conclusion that the thyroid is a toxin-destroying organ, the toxin being removed from the circulation and destroyed by its activity. He further believes that the toxin is elaborated in the intestinal canal from albuminous products. Animals deprived of their thyroid glands often manifest peculiar psychical symptoms, including hallucinations, character changes, loss of attention, etc. The tetanic attacks vary considerably in different animals; in some simple extensor cramp, in others clonic movements, etc., were observed. The psychic disturbances lasted from a few days to many weeks, and in some were distinctly periodical in character.

3. *Isolated Muscle Spasms.*—To be continued.

(1902, Vol. 21, No. 16, August 16.)

1. Pupillary Inertia in Accommodation and Convergence. J. STRASBURGER.
2. Multiple Neuritis and Basedow's Disease. T. DILLER.

3. Spinal Cord Degenerations in a Case of Old Arm Amputation. Ludwig Rosenberg.
4. The Tract "X" in the Lower Cervical Region. Purves Stewart.
5. Subcortical Origin of Isolated Muscle Spasms. A Contribution to the History of Quadrigeminal Tumors. Josef Sorgo.

1. *Pupillary Inertia.*—A report of two cases of hereditary syphilis and a case of multiple sclerosis, presenting a very marked slowing of the iris, contraction to light and accommodation. In the case of multiple sclerosis the dilation of the pupil on the affected side took 10-15-20 seconds, in the unaffected eye two to three seconds. In the first case of syphilis the right pupil was rigid; the left exhibited a typical slow reaction. The phenomena were present on both sides in the second case of syphilis.

2. *Multiple Neuritis and Basedow's Disease.*—An excellent report of a case of exophthalmic goiter in a woman of forty-six years, complicated by multiple neuritis which the author thinks was of toxic origin, and due to disturbance of the function of the thyroid gland.

3. *Spinal Cord Degenerations in Old Amputated Arm.*—The spinal cord of a fifty-two year old woman whose left arm had been amputated thirty years previously above the elbow, was carefully studied. The spinal cord in the lower cervical segments was smaller on the left side. The cell changes were marked. From the sixth cervical to the first dorsal segments the antero-external cell group had disappeared. In the eighth cervical and first dorsal the antero-median cell group had disappeared. The posterior horn was also atrophied. This would therefore give a localization for the forearm and hand muscles in the sixth cervical to the first dorsal segments of the cord inclusive.

4. *The Tract "X."*—A controversial discourse on the difference between the tract described by Stewart (tract X) and the tract described by Spiller. Stewart maintains that they are different fiber tracts.

5. *Isolated Muscle Cramp.*—To be continued.

McCarthy (Philadelphia).

Revue Neurologique.

(1902, Vol. 10, No. 10, May 30.)

1. Multiple Sclerosis with Transitory and Recurring Symptoms. Paralysis of Eye Movements in Right Binocular Vision and Later in Left Binocular Vision. Gilbert Ballet.
2. Two New Cases of Lesions Confined to the (Bourrelet) of the Corpus Callosum. J. Pelnar and Vl. Skalicka.
3. A New Method of Cerebral Measurement. Relative Atrophy of the Parietal Lobe by Comparison With the Frontal Lobe in Insanity. Maurice Dide and Louis Chenais.
4. A Parasite Found in the Blood of Epileptics. Bra.

1. *Multiple Sclerosis with Transitory and Recurring Symptoms.*—At present there is little difficulty, the author says, in the diagnosis of multiple sclerosis; nystagmus, vertigo, derangements of vision, scanning speech, intention tremors in head and arms, and spastic paraplegia are regarded as infallible evidences. Yet formerly the evolution of multiple sclerosis of which the present case is a good example, presented many difficulties which interfered with a successful diagnosis. The patient was a woman of thirty-eight years, married, who, although never robust, had enjoyed good health up to January, 1887, when after some preliminary symptoms of illness, she was attacked by binocular polyopia, which did not affect the vision when she looked to the extreme right or closed one eye. There was some weakness of the left side of the body,

sensibility was diminished and the pharyngeal and corneal reflexes were abolished. These phenomena appeared rapidly, then grew slowly less, disappearing altogether at the end of three weeks. After an interval of five years, in January, 1892, without any apparent exciting cause, she was seized with spasm of the left side of the face, followed by binocular diplopia involving the whole visual field, accompanied by intense vertigo and vomiting. The patient could not walk without assistance and complained of a sensation as if a ball were rolling about in her head. After about five days these phenomena disappeared with the exception of the last, and until January, 1893, she enjoyed fairly good health. At the end of that period on rising one morning she experienced nausea, vertigo and numbness with derangements of vision. Numbness of left side increased and there was difficulty in speaking and walking. Diplopia reappeared, both vertical and lateral, and there were spasmodic contractions of the left side of the face with right peripheral facial paralysis. This condition continued for about two years, but during the year 1895 the phenomena gradually subsided and but for a slight lateropulsion to the right and some difficulty in maintaining equilibrium the patient's health was satisfactory until the fifteenth of May, 1901. On that date vertigo and diplopia reappeared with paralysis associated with the lateral movements of the eyes to the right. These difficulties increased, walking became impossible, although muscular force was retained. The diplopia then gradually disappeared together with the other symptoms, although nystagmus persisted and the patient could not hold herself upright. Most of the reflexes were exaggerated, but there was no trace of facial paralysis. An examination of the eyes revealed nothing especially abnormal. The condition of the patient gradually improved, so that in a few weeks she was able to walk when assisted. Shortly after vertigo reasserted itself, but diplopia did not return. Whenever the patient attempted to walk she was seized with tremor, and although the right hand was quite steady the left was so tremulous as to be practically useless. This mode of evolution by successive attacks with suspension and intermission of symptoms is not new in the history of multiple sclerosis, but it rarely manifests itself in as striking characteristics as those of the case here presented, which is chiefly remarkable as a demonstration of the possibility and frequence of associated paralysis of the eyes in multiple sclerosis.

2. *Two New Cases of Lesions Confined to the (Bourrelet) of the Corpus Callosum.*—Two cases are here presented as a continuation of the researches of Marie regarding a peculiar lesion of the corpus callosum. In the cases now under consideration the lesion is confined to the (bourrelet) of the corpus callosum. As in the cases before considered, there was a brownish coloration extending in the second instance to the ependyma of the internal wall of the posterior cornua right and left. There are few similar instances reported in literature, and the importance of new researches is evident. In the first case here considered the lesion was confined to the lowest anterior portion of the (bourrelet). The "yellowish-gray" color was darker in the right half of the splenium. Laterally it extended only to the internal wall of the postcornu. Beside diffuse arteriosclerosis in the left hemisphere there was complete destruction of the third posterior of the hippocampus, of the lower lingual gyrus and the upper part of the fusiform gyrus. The occipital point, the calcarine fissure as well as the cuneus, were untouched. Another focus of softening was in the white matter of the left external wall of the posterior cornu. This lesion might be explained as a degeneration secondary to the lesion of the lingual and fusiform gyri, but there are certain cir-

cumstances which render this explanation unsatisfactory. There is a disagreement between the extent of the hemispherical lesion and that of the so-called degeneration of the splenium on the side opposite to the hemispherical lesion and a lack of coloring on the right surface of the fornix. Moreover no such explanation can possibly hold in the second case. Here the brownish color extended entirely over the lower half of the (bourrelet) of the corpus callosum, being darker in the right half of the splenium and reaching as far as the right internal wall of the postcornu to the left surface of the lower part of the fornix. There was diffuse arteriosclerosis in the hemispheres, but with the exception of little gaps in the left lenticular nucleus no localized lesion either of the cortex or the subcortex. The lesion of the splenium resembled that of the preceding case. The hypothesis of secondary degeneration was disposed of by the absence of extended destruction of the cuneus and of the posterior and internal parts of the temporo-occipital lobe. If this last case be compared with the second of those previously presented by M. Marie it is found that there was almost the same extent of the brownish coloring of the surface of the splenium and the same absence of lesions of the temporo-occipital lobe and the cuneus. In the first case here considered a small cystic cavity in the middle of the affected area of the splenium would seem to prove the primary lesion in the splenium.

3. *A New Method of Cerebral Measurement.*—While the ordinary method of weighing the different parts of the brain is useful in comparing the anatomical differences of its various parts, this plan is too arbitrary to be practical when considering the abnormal proportions of the brains of the insane with a view to obtaining measurements of the relations of the various lobes to each other. Moreover, an exact separation of the different parts is well nigh impossible, as one cannot always make arbitrary divisions. But there are certain points in the brain cortex whch are easily determined, and using these as starting points one can estimate roughly the area of the included surfaces, and make comparisons from one head to another. These measurements must be made as soon after death as is possible, in order to obtain the best results, as there is always a tendency of the convolutions to flatten, especially after the removal of the pia mater. For this reason many of the present measurements have been made without lifting the pia mater, and with a little practice the fissure of Rolando may be easily located. When the brain has been preserved in formalin the measurements are much easier, as the surface is then hardened. The details should be consulted in the original.

4. *A Parasite Found in the Blood of Epileptics.*—In a previous paper the announcement was made that the examination of the blood of seventy epileptics had revealed the presence of a parasite at certain periods of the disease. Cultivation of these parasites has now made a more complete account of them possible. The blood was examined just before the beginning of the attack and also during its course. Blood obtained after the paroxysm had passed gave varying results. In some cases no microorganisms could be detected, only the hyperleucocytosis observed by Kroumbmiller and a very active phagocytosis. The parasite is apparently present only at certain times. In blood obtained just before the paroxysmal attack small points, single or as diplococci were perceived moving about or united in masses vermicular in appearance. Examination of their structure showed that they are composed of grains of irregular shapes, and can readily assume an infinite variety of combinations. The number of those parasites found varies greatly in different individuals, but examination of a large number of healthy subjects failed to reveal

any similar micro-organism present in the blood. Cultures were made and from these intravenous injections upon rabbits. When administered in any large quantities the animals were either seized with convulsions terminating in death, or with simple rigidity, contractions, dilation of pupils, etc., and after the crisis emission of urine, relaxation of muscles, lowering of temperature, torpid condition for about an hour, and then a return to normal conditions. The varying severity of the attacks was probably due to the difference in the virulence of the infection. It is to be hoped that further experiment will enable the control of the amount administered. To what place shall these micro-organisms be assigned? The most recent experiments would suggest that they be considered as micrococci presenting themselves in the form of diplococci, and short chainlets capable of extension so as to take on the forms of coccobacilli, diplobacilli and streptobacilli. The frequent junction of the parasites and the hematin, the pallor and alterations of the parasites during the crisis period, the disappearance of a certain quantity of hemoglobin, the modifications in the coloring and consistency of paroxysmal blood which have been observed for a long time, altogether indicate that, like the agent of paludism, the parasite under consideration, lives at the expense of the normal elements of the blood.

Taking into consideration the fact that rare or absent during long intervals between seizures, the parasite shows itself in increasing quantities before the attack, lessening or even disappearing after the convulsive crisis, the epileptic attack may be regarded as a series of infections in which the convulsive crisis is parallel to the fever crisis of malaria. This also explains the intermittent periods. The securing of cultures and the results of inoculation leave little doubt, writes the author, that the micro-organisms here described are the pathological agents of epilepsy and that the symptom-complex which characterizes the disease is a result of the irritative action which they exercise over the different nerve centers through the medium of the circulation.

Revue Neurologique.

(1902, Vol. 10, No. 12, June 30.)

1. Progressive Muscular Atrophy (Aran-Duchenne type) with Intermittent and Temporary Contraction. HENRI MEUNIER.
2. The Sensibility of the Skeleton. MAX EGGER.
3. A Case of Pellagra Accompanied by Retraction of the Palmar Aponeurosis. PARHON and M. GOLDSTEIN.

1. *Progressive Muscular Atrophy.*—This author relates the history of a patient aged forty-three years, a hotel porter. His antecedents gave no reason to suspect syphilitic affection or the result of alcoholic overindulgence. The first indication of the presence of the disease was a gradually increasing weakness of the right hand, which soon made writing impossible. The paralysis gradually progressed up the arm to the shoulder, but was not accompanied by any pain, except occasionally involuntary spasms with sensation of heat and smarting. From these symptoms the diagnosis of syringomyelia was made and electrical treatment applied. The paralysis extended to the left hand and then to the right leg. The examination made at this point revealed no loss of the different sensibilities. The right arm was limp and emaciated, the fingers almost powerless, all movement difficult or impossible. The left hand only was affected and that in a less degree than the right. The legs were weak, often contracting during the night, and straightened with difficulty on arising, so that for the first hours of the day the patient was

unable to place the sole of the foot upon the floor and was compelled to walk on his toes. There was no tremor, but he had fibrillary spasms which were plainly visible. The application of electricity gave the following general results: faradic excitability was less in all the muscles of the right side compared with those of the left; voltaic excitability seemed generally diminished. The two patellar reflexes were perfectly normal, and the ulnar equally so. The left wrist reflex was normal but the right inappreciable. Apart from the muscular atrophy itself there were no trophic disturbances. There were no vasomotor disturbances, yet the patient was subject to excessive sweating, occurring almost without an exciting cause and often accompanied by a sensation of heat in the right shoulder. The author sums up these symptoms, which in his opinion give a clear case of Aran-Duchenne's disease, and by a careful comparison eliminates the first diagnosis of syringomyelia, and several others that might at first seem to be indicated. The phenomena mentioned of difficulty in extending the leg when it contracted during the night, and the inability to place the sole of the foot on the ground would seem to be a new one in the history of this disease, although instances somewhat similar have been recorded. It is somewhat difficult of explanation, for it is evidently not a sclerotic retraction as it occurred before the atrophy of the limb. It is the author's opinion that it was produced by a slight extension of the lateral cord in a certain part of his dorsolumbar medulla (pyramidal tract) in the process of sclerosis.

2. *The Sensibility of the Skeleton.*—Our knowledge of the degree of sensibility of which the bones are capable is as yet very limited. The usual method of percussion can only be satisfactorily employed when the intervening skin and muscles are entirely anesthetic. This author has previously demonstrated that the molecular vibrations of a tuning fork are perceived by the bones when the intervening tissue is unaffected. In some cases of tabes the bones have been found to be without sensibility when it was normal in the skin and muscles, and in one case of Pott's disease with absolute anesthesia in the three modes of superficial sensation in the lower limbs, the vibrations of the tuning-fork were plainly felt by the bones. According to the opinion of this author the perception of segmentary attitudes of the limbs is not due to muscular sensibility, but he admits the question is not definitely settled. He has, however, often observed cases where there was loss of perception of segmentary attitudes, but the patient still retained the sensation of vibration upon the bones. The examination of a case of lateral amyotrophic sclerosis in which the two upper extremities were affected by excessive muscular atrophy showed that vibration just perceptible in a normal individual was quite as plainly perceived in the atrophied limbs of these patients. Various experiments have gone far to prove that the sensation of vibration is felt only by that part of the skeleton upon which the tuning fork is directly placed. Anesthesia of some parts often exists when other parts are unaffected. In cases of painless spontaneous fractures in tabes and other affections these fractures were anesthetic to vibrations, although in one case the sensation was found to be not only present but exaggerated.

3. *Pellagra Accompanied by Retraction of the Palmar Aponeurosis.*—These authors relate the history of the case of a man of twenty-seven years. At the age of six years he had been subject to paroxysms during the night, the precise nature of which could not be learned. These had ceased about his sixteenth year without treatment, which was thought would hardly admit the hypothesis that they were epileptic. The patient gave no evidence of hysteria. Perhaps the result of nightmare following

a fright. A little later he was often attacked by severe cramps in the left leg, and following these the characteristic pellagris erythema put in an appearance. There was also diarrhea for three years often accompanied by violent abdominal pains, and gradually the left hand became deformed and useless. The physical examination showed a tendency to carry the head a little to the left, but it was probably due to habit as the patient could place it in a normal position without difficulty. The left forearm was slightly drawn out of position and the fingers of the left hand much contracted, so that an attempt to straighten them caused pain to the patient. The integuments of the two hands were of a purplish color on both surfaces and always covered with sweat. This color was present a little less deep upon the thighs and legs. Sensations were all normal, although vision was slightly affected, a not uncommon occurrence in pellagra. Most of the reflexes were normally present except in the left forearm and hand. The memory appeared to be considerably enfeebled. The power of abstraction and mental representation was greatly altered. The patient believed himself persecuted by his relatives. There was also a tendency toward megalomania. It is the opinion of the authors that the deformity of the left hand was due to a retraction of the palmar aponeurosis, as the fact that the patient could easily move the second and third phalanges of the middle and right little finger removed the possibility of contraction or paralysis. In their opinion it was a trophic affection due to an alteration in the nervous system, although they were able only to conjecture the location of the lesion which caused it. It may possibly depend upon a lesion of the peripheral nerves, or upon alterations of the cells of the gray substance of the periependyma. JELLIFFE.

Archiv. für Psychiatrie und Nervenkrankheiten.
(1902, Vol. 36, Part I.)

1. Recent and Old Investigation of the Brain. EDWARD HITZIG.
2. A Contribution to the Pathology of Porencephaly. H. ZINGERLE.
3. Pachymeningitis Cervicalis Hypertrophica and Pachymeningitis Interna Hemorrhagica with Chronic Progressive Dementia in the Young. M. PROBST.
4. A Contribution to the Etiology of the Periodical Psychoses. CLEMENS NEISSER.
5. Metastatic Abscesses in the Central Nervous System. R. CASSIRER.
6. Diffuse Hemorrhagic Encephalo-myelo-meningitis. MARTIN BARTELS.
7. Central Neurofibromatosis and Tumors of the Cerebello-pontine Angle. HENNEBERG and MAX KOCH.

1. *Recent and Old Brain Researches.*—A detailed study of the methods and results of brain investigations and experiments. Too extensive and detailed to admit of a satisfactory short summary, but withal an important series of contributions.

2. *Contribution to the Pathology of Porencephaly.*—The brain was obtained from a laboring man who, during life, showed no evidences of paralysis and whose death was caused by an injury. On the outer surface of the left hemisphere there is an extensive defect (*einsenkung*) including the greater portion of the central convolutions, the supra-marginal gyrus and the posterior portion of the second and third frontal convolutions, the area corresponding to the second and third branches of the Sylvian artery. Over the depression the meninges were thickened and the cortex atrophic (microgyra). The deficiency exists chiefly in the subcortical white substance, which is replaced by a dense scar tissue. A

few scattered areas are present in the cortex. In the opposite hemisphere, symmetrically situated, was a small sub-cortical cyst. The author, who had made a very elaborate study of the case, believes the condition to have been of pathological origin, occurring during intrauterine life (*i.e.*, not a developmental defect). Kundrath suggests that such cases are of vascular origin, the vessels not being completely occluded. This forms an impediment to the circulation, which affects chiefly the deeper structures, because of the greater blood pressure required and the meager possibilities of collateral circulation. The advanced state of fissuration in the diseased area renders the later part of fetal life as the probable time of onset. The other portions of the affected hemisphere and that part of the opposite hemisphere, corresponding to the defect had undergone a considerable hypertrophy. This curious vicarious growth may compensate for the loss of substance incurred and explain the absence of symptoms.

3. *Pachymeningitis Cervicalis Hypertrophica and Dementia in the* Young.—Case I. Age fifteen years, syphilitic taint (?). When thirteen years old a severe occipital trauma occurred, followed by convulsions, later headache and vertigo. Convulsions recurred. Paresthesia present. Progressed slowly to dementia. The pupils were unequal, stiffness of neck, incontinence, atrophies in the face and extremities (degeneration reactions), legs paralyzed, slight claw-hand. Objective sensory disturbances were present; blind and deaf. Discs normal, no fever throughout, the pulse slow. Autopsy revealed a pachymeningitis interna hemorrhagica with atrophy of the subjacent cortex. Pachymeningitis cervicalis hypertrophica with cervical myelitis. Lumbar and sacral cord free. Degeneration of the pyramidal tracts in the cord only.

Case II. Age thirteen years, no hereditary taint. Rickets. Began with loss of memory, progressing slowly to dementia. Pupils unequal, nystagmus. Optic atrophy due to a chronic choroiditis. Jacksonian epilepsy in both sides. Atrophy of the small muscles of the hand (reaction of degeneration). Stiffness of the neck. Afebrile throughout.

Autopsy—Atrophy and chromatolysis of the ganglion cells of the cortex. No pachymeningitis interna. Well marked pachymeningitis cervicalis hypertrophica but without myelitis or degeneration of the pyramidal tracts. Similar in the two cases were: the age of two patients, the complete dementia, the afebrile course and cervical pachymeningitis. In both cases the process began in the brain, only after a perceptible interval extending to the cord. Thus supporting the theory of Köffen-Weiting that the cervical pachymeningitis is but the intimation of a meningoencephalitis.

4. *Contribution to the Etiology of Periodical Psychoses.*—Clemens Neisser says the important etiological factors in the production of the periodical insanities are: Heredity, trauma and organic cerebral disease. Those originating from organic disease showing a tendency to dementia. He reports a case following an apoplectic seizure, of the typical circular type. Mention is made of a case in which a stroke of lightning was the only demonstrable cause. Also a case following a trauma in which group of cases the prognosis is especially favorable. A wide mobile pupil during the interval is thought to indicate a persistence of the malady, although the patient may appear quite well.

5. *Metastatic Abscesses in the Central Nervous System.*—The author reports two cases of abscess formation in the pons and spinal cord respectively.

Case I. Onset with headache and fever of a pronounced hectic type. Five days later paresthesia and objective sensory disturbances on the left

side. Paralysis of the right abducens and seventh nerves. Keratitis neuro-paralytica in the right side. No paralysis of the extremities. Later stupor and optic neuritis. Death in coma twenty-one days after the onset.

At the autopsy a solitary abscess occupying the right half of the pons was found. Also multiple abscesses in the liver and lungs. The only explanation of the bacterial infection was found in a small ulcer in the vermiform appendix caused by a fish-bone. In the literature the author has collected thirteen reliable cases of abscess of the pons and medulla oblongata. Of these three were confined to the medulla. Abscesses in this region are nearly always metastatic. Some of the sources of infection are bronchiectasis, prostatic abscess, middle ear disease. Trauma, carious process in the bones of the skull and suppuration of the accessory sinuses, so commonly the cause of abscess formation in the cerebrum and cerebellum, is rarely affective in this locality, due to the anatomical relations and protected situation of the parts. The striking immunity of the brain stem to abscess formation of metastatic origin, has led some writers to suggest a chemical peculiarity as the possible explanation. Martins would seek the explanation in the general laws governing non-septic embolism, so common in the middle cerebral and so rare in the vertebrals or basilar. The spinal cord is likewise rarely the seat of abscess formation. Chiari was able to collect fourteen cases.

Case II. In the cord of a woman who had presented the symptoms of an advanced syringomyelia, myriads of minute abscesses were found, the largest scarcely visible to the naked eye. These miliary accumulations of normal cells were sharply circumscribed, and were found chiefly in the lumbar region most numerous in the gray matter and usually perivascular. The source of infection had been a fresh endocarditis. There were also infarcts in the spleen, lungs and kidneys.

6. *Diffuse Hemorrhagic Encephalo-myelo-meningitis.*—Martin Bartels reports the case presenting a variegated picture of cerebro-spinal disease. Headache, stupor, convulsions, vertigo, transient attacks of aphasia and paralysis. Optic neuritis. Hyperesthesia of the special senses. Dermographia and transient flushings of the face and body. Slight albuminuria. Histological examination disclosed a remarkable process, affecting the veins of the central nervous system. A periphlebitis, phlebitis and a large celled endophlebitis, analogous to Heubner's endarteritis. The arteries apart from the periarteritis presented no change. Nowhere was an endarteritis demonstrable. The intense proliferation produced nodular and semilunar elevations, sometimes concentric. The lumen was not obliterated and nowhere could thrombi be demonstrated. Giant cells were found occasionally, but neither gummata, tubercles nor areas of necrosis. The cord was the seat of diffuse degenerative changes. Small hemorrhages had occurred in the basal ganglia and in the cortex, but none in the cord. The degeneration in the brain was very slight. The layers of the meninges were diffusely infiltrated with round cells, as were also the perivascular lymph spaces. The kidneys did not show these changes. The other organs were not subjected to microscopical examination. These changes were attributed to syphilis and were thought to represent an early stage of the cerebrospinal affection. Mention is made of Reider's investigations, who claims that the first effect of syphilis is an endophlebitis. The large-celled endophlebitis of the umbilical vein in fetal syphilis has been confirmed by various observers.

7. *Central Neurofibromatosis and Tumors of the Cerebello-pontine Angle.*—The authors, Henneberg and Koch, present complete studies of two cases of bilateral, symmetrically situated tumors of the cerebello-

pontine angle. One was the central localization of a generalized neurofibromatosis, the other recurring in conjunction with multiple fibromata of the meninges. In both cases, the pontine tumors were intimately connected with the acoustic nerves and attained the size of horse-chestnuts. Attention is called to the frequency of tumors arising in this locality, springing most commonly from the eighth nerve, but also from the fifth, seventh and other bulbar nerves. Histologically the neurofibromata are most frequent, but gliomata also occur. Normally elongations of glia may be traced for about one cm. in the extra-medullary portion of the nerve-roots. The growth fills in the cerebello-pontine angle (*cerebellar-brüken winkel*) and produces a more or less characteristic syndrome. The most important and most constant symptom is an auditory paresthesia or paralysis, but this may occur late. Headache is usually occipital, but may be frontal, and rarely crossed frontal headache has been observed. The latter due to irritation of an anterior cerebellar peduncle. Common symptoms are facial paralysis, trigeminal neuralgia, cerebellar ataxia, nystagmus, rarely paralysis of the associated ocular movements. Peculiar forms of dysarthria and vasomotor disturbances have also been noted. Hemiplegia may be absent, but if present, is usually on the same side as the tumor (centro-lateral pressure against the base of the skull). It has, however, been observed on the opposite side. Tumors in this locality may simulate some of the following clinical pictures: bulbar paralysis, Menière's disease, paraplegia, aneurism, lues cerebri, and hysteria in an initial stage. J. RAMSAY HUNT (New York).

Rivista di Patologia Nervosa e Mentale.

(1902, Vol. 7, fasc. 8, August.

1. Upon Indirect Secondary Atrophy of the Nervous Elements. E. TANZI.
2. Polyclonia in Dementia Paralytica. R. LAMBRANZI.

1. *Secondary Atrophy of Nerve Elements.*—The author reviews the literature of this subject and contributes a description of his findings in the experimental study of neuronic atrophy from functional inertia through suppression of a given function from birth. Enucleation of the eyes was practised in newborn rabbits and dogs; the animals being killed from six months to a year after operation, and the condition of the visual center studied macroscopically and microscopically. The findings are minutely described in these as well as in the case of an anophthalmic dog. The salient points in the conclusions drawn are that lesions of the nervous system of the newborn animal induce complete degeneration solely in those neurones which are directly injured. Those neurones which are simply in relation with the injured, but are not themselves involved, undergo a process of atrophy but are never entirely destroyed. As suppression of functional stimulus is incapable of causing disappearance of cells, but can only render them atrophic or arrest their development, it may be inferred that every nerve-cell has hereditary power to attain a certain degree of development and to this hereditary development is added that which is derived from its anatomical connection and consequent functional activity. Such complement of the ontogenetic development is greater in the higher order of animals which have a more "plastic" cerebral cortex and are capable of greater development. It may be inferred also that the higher orders of nervous centers are the most susceptible to the influence of suppression of functional stimuli.

2. *Polyclonia in Dementia Paralytica.*—This article follows a line of argument presented in a previous work, tending to show that myoclonia

has its origin in a deviation from the normal in the cerebral cortex. A case reported by the author in which myoclonia was the initial symptom, followed by exaggerated patellar reflex, myosis, spastic gait and psychic dulness with gradual development of a typical dementia paralytica, lends support to the author's theory of the cerebral origin of polyclonia.

(1902, Vol. 7, fasc. 9, September.)

1. Upon the Presence of Neuroglia in the Choroid Plexuses. C. Católa.
2. Changes in the Nerve-cells in Acute and Chronic Iodoform Poisoning. R. Giani and E. Ligorio.
3. Contribution to the Study of the Hygric Illusion. E. Ravenna and T. Montagnini.
4. The Fasciculus of Pick. F. Ugolotti.

1. *Neuroglia in the Choroid Plexuses.*—In a former article, the author has published the report of a glioma at the level of the choroid plexuses of the fourth ventricle. To prove the theory then advanced as to its development from neuroglia normally existing in the choroid plexuses, he has studied numerous microscopical specimens of the choroids and has determined the existence of a delicate layer of neuroglia beneath the ependymal layer in nearly every instance.

2. *Changes in the Nerve-cell in Iodoform Poisoning.*—A report of the condition found in animals after death from acute poisoning by means of large injections of iodoform with glycerine or olive oil, and from chronic poisoning by means of small doses repeated at intervals of three or four days. For full details the reader is referred to the original article; suffice it to say that no part of the cerebrospinal system was exempt from the noxious influence of the drug; cellular degeneration and chromatolysis being widespread.

3. *Contribution to the Study of the Hygric Illusion.*—An interesting case, in which hygric illusions were a prominent characteristic, is reported by the authors, who discuss the center to which such illusions are referred and, from the findings at autopsy in the present case, consisting in various deviations from the normal in the hippocampal convolution, contribute further support to the hypothesis that hygric illusions may be referred to that area.

4. The Fasciculus of Pick.—This anomalous fasciculus has been found in the medulla oblongata by the author in three cases out of twenty-six. Its point of greatest size was found at a level with the decussation of the pyramidal tracts, from which point it decreased toward the inferior portion of the bulb. Ugolotti leans to the belief of Hoche that the fibers of the fasciculus are derived from the pyramidal tracts. The question of their distribution is taken up, but no definite conclusion is reached.

Fielding (New York).

Nouvelle Iconographie de la Salpêtrière.

(15th Year, No. 3, May-June, 1902.)

1. Three Cases of Cerebral Neoplasm. G. Ballet and Armond-Delille.
2. Description of a Case of a Rare Monstrosity of a Face and Head. Haushalter and Briquel.
3. Psychasthénic Syndrome of Akathesia. F. Raymond and Pierre Janet.
4. Biological Life of a Xypophagus. Vaschide and Vurpas.
5. A Case of Hemimelia of the Right Lower Extremity, Studied by Means of a Radiograph. Infroit and Heitz.
6. A Case of Eunuchism of a Family Type. P. Sainton.
7. Vesical Calculi in Holland. Bolk and Mayet.

1. *Cerebral Neoplasm.*—An account of three cases of cerebral tumor with autopsy findings, which present some unusual features.

Case I—Man, 53 years old; headache, right hemiparesis, slight speech trouble, not, however, aphasic; coma, deaf. Autopsy: gliomatous tumor about the size of a walnut, located on the posterior part of the third frontal convolution of the left hemisphere. It is interesting in this case to note that, although the tumor was of large size and compressed the foot of the third frontal left, it did not extend far beyond the motor area.

Case II—Appearance at ten years with epileptiform attacks during two years following. Apparent return of health, then headache, with trophic symptoms located in the head, especially in the left parietal region, facial hemiparesis on the right side. Renewal of epileptiform attacks, vomiting, incomplete right hemiplegia, without aphasia, prolonged terminal period, with stupor and death. Enormous sarcoma, the size of an orange, compressing the left frontal lobe. The remarkable toleration which the brain exhibited for a growth of this size is worthy of attention. Although the tumor was so large that it caused direct trophic changes on the skin, hair, and skull, lying directly over it, by pressure, yet, with the exception of a slight facial paralysis, no grave motor symptoms were noted. In this case also the absence of aphasia, in spite of the pressure on both Broca's convolutions, is noted. This case would tend to show that the gray substance of a convolution can be compressed to a great degree without its function being markedly affected, provided always that the increase in pressure proceeds slowly and progressively.

Case III—Following a trauma of the head in a man 19 years of age, rapid development of paralysis of the intrinsic ocular muscles and Jacksonian epilepsy, progressive; difficulty in swallowing, profound torpor, death. Diffuse gliomatous infiltration, predominating on the anterior two-thirds of the convolution of the corpus callosum of the right hemisphere. This case shows the following points of interest: Diffuse gliomatosis; traumatic origin; symptoms suggesting in the beginning a bulbar affection.

2. *Rare Monstrosity of Face and Head.*—An account of a monster showing an arrested development of the face, deformity in the hands, feet, brain and skull. The cause of all of these defects of the brain was probably a hemilateral hydrocephalus. The article is illustrated with clear photographs and contains exact descriptions and measurement of the subject.

3. *Psychasthenic Syndrome of Akathesia.*—Haskowec of Prague reported to the Neurological Society two cases of a peculiar nervous malady which he called Akathesia, or the impossibility of seating oneself. This paper is an account of such a case, observed at the Salpêtrière. Man, 42 years old. When he sits down in a chair he becomes greatly embarrassed and shows evidence of great mental and physical suffering. He twists his body about, alternately flexing and extending his legs, allows his head to fall on the left shoulder, and at the same time breaks into a sweat and has attacks of palpitation of the heart. His expression shows great pain, terror and suffering. At length, unable to hold himself any longer in the chair, he rises quickly and the attack ceases completely. Haskowec considered that this syndrome bore some resemblance to the astasia-abasia of hysterical subjects, but Raymond believes that it is of different type entirely, and for the following reasons: (1) The patient is able to sit down and to remain seated, the symptoms only beginning after the lapse of a certain length of time. (2) Even

when the attack begins, it is only necessary to question the patient or to speak to him, when he becomes quiet. (3) He does not fall from his chair. (4) The cause of this attack is not due to an incapacity to remain seated, but it is an agony and suffering which develop when he is seated. The author believes that the syndrome of akathesia is of psychasthenic origin and is really a sort of profession aboulia with attacks of anxiousness. A somewhat similar case is related in the same article.

4. *Biological Life of a Xypophagus.*—This is a very interesting study from the physiological point of view of a Chinese xypophagus, exhibited by Barnum & Bailey at Paris under the name of the "Chinese Brothers." They are fifteen years of age and are united by a band at the xyphoid region, which, by an X-ray picture, is shown to be composed of cartilage. A careful anatomical description, illustrated by photographs and containing also accurate measurements, is included in the article. The investigation consists of studies of the pulse, respiration, muscular force, etc. The following conclusions, in brief, are noted: (1) Each subject at the same time leads a biological life with an individual life. This individual life has very definite limits from the standpoints of circulation, respiration, sensation, and muscular activity. (2) One of them is more vigorous from the point of view of muscular activity than the other. (3) In spite of the existence of these two lives, each of which reacts in a great measure to their individual laws, there is a harmonized biological life which has its origin deeply rooted in the two organisms. This life is the resultant of the two others and reduced itself to a condition of well equilibriated automatism.

5. *Hemimelia of the Right Lower Extremity.*—An article, illustrated by photograph and radiograph, of a malformation of the inferior right extremity of a woman, fifty-eight years of age.

6. *Eunuchism of a Family Type.*—The term, Eunuchoid, is given to those individuals who in many respects present characteristics commonly found in eunuchs, due to an atrophy of the testicle, resulting from localization of an infection of the testicle or by the arrested development of that organ. This article is an account of an individual who showed the personal and family characteristics from this point of view. Several members of his family, in different generations, have had the same peculiarity. SIDNEY I. SCHWAB.

(1902, No. 4, July-August.)

1. Pure Verbal Blindness. E. BRISSAUD.
2. Hallucinations of Hearing in a General Paralytic. SÉRIEUX and MIGNOT.
3. Study of the Root and Cell Lesions of Tabes. A. THOMAS and HAUSER.
4. Dementia Præcox and Katatonia. J. SÉGLAS.
5. Multiple Exostosis with a Tendency to Suppuration. LAUNOIS and ROY.
6. Psychiatry in the Japanese Theater. GEYER.

1. *Verbal Blindness.*—A case of verbal blindness caused by softening in the left calcarine region with degeneration of the splenium and the tapetum on the right side. A man, fifty-seven years old, entered the hospital with a history of blindness, which had existed for several months. On examination it was found that he was not blind, that he could write, but that he was incapable of reading even his own signature. This symptom can with justice be called verbal blindness. Patient died in twenty-four hours. The localization of the lesion of softening in the occipital

zone was as follows: It occupied the posterior extremity of the internal surface of the left hemisphere. It was the so-called yellow softening, resulting from a total ischemia of at least several weeks' duration. The cortex and the adjacent parts are affected and there is some meningitic adhesion. The calcarine artery is obliterated, causing a necrosis, the focus of which includes almost the whole of the cuneus. The only parts not touched are, first, the occipital pole itself; second, the anterior superior border. The tapetum and the optic fibers, however, have completely disappeared. The degeneration of the tapetum is the necessary consequence of the lesion of the gyrus lingualis. It extends to the splenium of the corpus callosum.

2. *Hallucinations of Hearing in General Paralysis.*—The study of the sensory forms of dementia paralytica have up to this time been singularly neglected in spite of their importance and frequency. The authors have recently observed a case in which the excitation or paralysis of certain sensory centers have produced symptoms which dominated the whole clinical picture. A man forty-one years old, in whom a delirium of persecution and of grandeur has become systematized under the influence of sensory symptoms with hallucinations of hearing, smell, taste and general sensibility, forms the subject of this paper. Several times his sensory delirium has been interrupted by epileptiform attacks. At the autopsy a diffuse meningo-encephalitis was found with foci sharply circumscribed and located on the posterior aspect of the brain. The anterior aspect of the brain was relatively free from lesions. A knowledge of this clinical and pathological fact is of importance from the diagnostic point of view. The existence of a delirium based upon hallucinations and the observation of symptoms such as verbal blindness and sensory aphasia, usually caused by focal lesions (softening, etc.) should not necessarily carry with them the usual diagnosis of diffuse meningo-encephalitis, but should suggest the sensory form of dementia paralytica, which depends upon the existence of foci of meningo-encephalitis, more or less circumscribed and limited to the posterior portion of the brain.

3. Continued article.

4. *Katatonia and Dementia Praecox.*—In 1874 Kahlbaum described, under the name of katatonia, a symptom-complex with a cyclic evolution, the chief symptoms of which were a spasmodic motor trouble of the nervous system. (Spannungs Irrsein.) Since then katatonia has been the subject of numerous investigations, until Kraepelin brought forward his well-known theory that hebephrenia and katatonia are not separate conditions, but are phases of one morbid state to which he gave the name dementia præcox. The following are the chief general conclusions which have been advanced by Finzi and Vedrani: 1. The syndrome of katatonia is found more or less pronounced in many forms of mental disease. 2. It never forms a definite clinical picture. It is not a complete disease, and occupies only certain periods of the morbid process. 3. It is found most completely and most permanently developed in cases of dementia praecox, which have the closest relation to hebephrenia. The principal symptoms which, since the time of Kahlbaum, are considered under the name of katatonia, are stereotypy of attitude, words, acts, strange and absurd, and a tendency towards cataleptic immobility, with a tension of the muscles almost tetanic, and a rigidity more or less pronounced and permanent. This symptom-complex being present in repose, becomes exaggerated in the resistance opposed by the patient to passive movements, a resistance which explains the patient's refusal to take food and his absolute mutism. Kahlbaum gave the name Negativismus to this group of

phenomena of opposition. To illustrate these symptoms and the foregoing conclusions, the author describes three typical cases of katatonia.

5. *Multiple Exostoses.*—Numerous cases of multiple exostoses have been published in the last two years. In a recent article, Auvray and Guillain have noted all the known facts, together with two new cases of this condition. The objective characteristics of this disease are the symmetrical localization of the exostoses in the extremities of the long bones, grouped usually about the epiphyseal cartilages and developing at the same time with the skeletal growth. They can remain unnoticed for a long time, can persist indefinitely, and even sometimes showing a tendency to disappear. They are usually painless, but are capable of causing symptoms by pressure. The case which is here reported, as an illustration of this disease, is a man thirty years old, with multiple exostoses, some of which are accompanied by suppuration. In addition, the patient shows a symptom of syringomyelia, a Morvan hand, and sensory symptoms, characterized by hemianesthesia and a thermo-anesthesia of the left side. The exostoses are present in various parts of the body, which a diagram and a radiograph well illustrate. The author concludes as follows: 1. The pathogenesis of this disease is not known. 2. Perhaps it is dependent upon an affection of the nervous system not yet localized (gray matter of the cord). 3. The suppuration in this case can be explained by the presence of tuberculosis. S. I. Schwab (St. Louis).

Archives de Neurologie.

(1902, Vol. 13, No. 78, June.)

1. Three Cases of Hemianopsia. F. Raymond.
2. Contribution to the Psychology of the Genesis of Psychomotor Hallucination. N. Vaschide and Cl. Vurpas.
3. Observations in Five Cases of Conjugal General Paralysis. P. Keraval and G. Raviart.

1. *Hemianopsia.*—Using three cases as examples Raymond offers a clinical lecture on hemianopsia in which he describes the symptoms and pathological lesions in a very interesting and clear manner.

2. *Psychology of Genesis of Psychomotor Hallucinations.*—A case of psychomotor hallucinations. The patient, a woman forty-three years old, hears voices speaking within herself and, without trying to localize themselves in any part of the body, affirms that they speak to her. She moves her lips to utter the ideas of persons that talk within her and answers them. Thus she carries on a dialogue. At times she believes what she says and writes her own thoughts, and then at times she thinks they come from elsewhere. Therefore in this case the so-called psychomotor hallucinations are only the ideas of the patient of which she is conscious by exaggerated mental introspection. Ignorant of psychological laws of association, she is astonished at possession of thoughts which she knows to be contrary to her sentiments and to her moral and intellectual personality, and at once ascribes them to an exogenous origin. When the mental image produced by these thoughts is strongly enough developed, she is led to a motor reaction and articulates these thoughts. Introspection plays the principal rôle. The authors do not wish, they say, to base a theory of psychomotor hallucinations which might arise under other conditions, on an isolated case. They wish to insist only on the genesis and mechanism of the case observed. The psychological examination ought to give most important details of this morbid symptom-complex, but the examination must be complete in order that the pathogenesis be of any value. The method of considering the syndrome by itself, in its

characteristic traits, and explaining in a way to satisfy the mind, either by psychological facts, or those of psychiatry, or by other known, accepted and related phenomena, is a dangerous one. It isolates artificially the syndrome or series of facts from the unity of conditions to which it is intimately and indissolubly bound. What is needed is a complete detailed psychological examination. It is necessary to have sounded and penetrated to the depths of a subject's character before beginning the psychological experimental investigation. The observations of the subjects confirm largely the ideas held concerning the hallucinations commonly known as psychomotor. But in the study of the mental state of L—, independent of the physiological conditions, they think they can describe the mechanism and genesis even, of these hallucinations. The case is typical and therefore they have chosen it, and besides, the explanation can be supplemented by a number of indispensable elements furnished by observation. The subject under consideration does not present a case of well-defined symptoms, and would not enter into the category of morbid affections, if she had the necessary discernment between the different mental states of her distraction. There is a current opinion that the entire mental activity manifests itself in the form of a subconscious language. Subconscious language is, so to speak, the sanctuary of articulate speech, and hence of the *ego*—the conscious self—which enters into relation with the surrounding world, and which formulates acts and coördinates syllogisms. A deaf-mute has unspoken language extremely rich, although unable to articulate. It is rare that an individual can classify his thoughts or reconsider past impressions without the aid of this interior language.

The sole desire here is, without entering into discussion, to call attention to the influence in morbid psychology of this language on the systematization and origin of psychopathic trouble. This patient no doubt was ignorant of the subconscious life, and was surprised to find a physical and mental life whose origin, significance and purpose she could not understand. All this subconscious mentality furnished her the elements of hallucination by introspection. She constantly hears words as if whispered, and which persist in crossing her opinions. The attention is fixed on this phenomenon, and not finding any means to explain it, she ponders more and more over it till, in time, she comes to think that strange voices are talking and insinuating themselves into her thoughts.

What happens to L— has so far no pathological significance unless it is the adaptation of too sustained intellectual efforts, and the hasty but logical interpretation of facts which escape her in real consciousness. Each normal man may be appalled at the greatness and complexity of psychological phenomena. Many cannot fix the attention sufficiently to know this territory, and those who can are distinguished from those who present pathological trouble, by the fact that they represent the interior language as their own intellectual state. L— dissociates very minutely the articulated language from the subconscious, and here begins the pathological condition. She has not the judgment to see that the subconscious states are part of her ego, and by and by she attributes the "voices" to personalities identical with her former sympathies and antipathies, and suddenly begins to fight with illusory personages. Subconscious language is, therefore, the essential element of a number of psychomotor hallucinations, and perhaps of all. Instead of insisting upon accidental dynamic cerebral explosions or cortical irritation, one must study, for interpretation of this phenomenon, the hallucinations of subconscious language.

To sum up very briefly, psychomotor hallucinations can easily re-

ceive a psychological explanation on the ground of pathological introspection and attention to subconscious language.

3. *Conjugal General Paralysis.*—Fives cases are reported in which man and wife have developed general paresis. The etiological factor was not always evident, but in two of the instances syphilis played the leading rôle. In one family alcoholism is the only known cause, while in the two remaining cases cranial injury and heredity appear as causative agents in the husband, and grief and sorrow induce the disease in the wife. Regarding these latter cases, there is room for much skepticism. In three of the families the husband died first and in the other two they suppose the wife to have succumbed first. One woman developed tabes many years before there were any signs of general paresis. There was nothing of special note in the clinical pictures presented.

(1902, Vol. 14, No. 79, July.)

1. New Contribution to the Study of Vertiginous Epilepsy and Its Treatment by Bromated Camphor. Bourneville and Ambard.
2. Contribution to the Study of the Action of Valerian and Valerianates. Ch. Féré.
3. A Parasite in the Blood of Epileptics. M. M. Bra.

1. *Treatment of Vertiginous Epilepsy.*—Bourneville, as the result of years of treatment of epilepsy at Bicêtre, advises the administration of monobromated camphor, in gradually increasing amounts. When the dose has reached to five times the initial dose, it is gradually diminished. He does not give the amount of camphor used at the start. He reports the cure of three cases of the vertiginous form by this method.

2. *Action of Valerian and Valerianates.*—In small doses they excite voluntary motion. In stronger doses they produce depression. In any strength they cause diminution of resistance to fatigue. There is thus produced an inverse effect according to the strength of drug used. The physiological action as proved by experiment also differs in an interesting way from the antispasmodic and sedative effect which empiricism attributes to the drugs.

3. See abstract from *Revue Neurologique* in this number.

(1902, Vol. 14, No. 80, August.)

1. A Case of Epithelioid Papilloma of the Red Nucleus. Contribution to the Study of the Functions of the Red Nucleus. F. Raymond and R. Cestan.
2. Infantile Tremors and Congenital Nystagmus. An Attempt at Classification. Lenoble and Aubinean.
3. Notes Upon a Case of Epileptic Delirium. A. Petit.
4. The Hysteria of Saint-Theresa. Roubv.

1. *Papilloma of the Red Nucleus.*—The case reported is of interest from both a pathological and a clinical standpoint. The tumor presents microscopically a close resemblance to the metastases formed from a growth primary in the intestinal canal. However, after a most careful search at the autopsy no primary focus was found, and the reporters have decided that the neoplasm is primary in the brain, and originates probably from the pia mater covering the crura cerebri; that it is of the

nature of an endothelioma and akin to the "cerebral papilloma" of Rindfleisch, and the epithelioid endothelioma of Triegler. Drawings of the microscopical appearance of the tumor are given. The growth destroyed the left red nucleus completely, the inner half of the right red nucleus, and both third nerve nuclei. The crura were unaffected. The Marchi method demonstrated some degeneration of fibers in the right superior cerebellar peduncle; a small number of degenerated fibers on either side of the median line in the medulla just anterior to the posterior longitudinal bundle; a few in fibers to the island of Reil; but none in the pyramidal tracts and no evidence of a red nucleus-spinal tract. The clinical picture presented was as follows: the symptom-complex of Weber, in that there was a paralysis of the left oculo-motor nerve (primarily) associated with motor disturbances of arm and leg of the right side of the body, which did not consist in a true paralysis, but merely slight paresis accompanied by incoördination and ataxia. There was pronounced titubation and the speech was affected by disarthria. The reflexes were exaggerated. From a study of the case in its clinical and pathological aspects, Raymond and Cestan conclude that the red nucleus exercises an influence over the tonus of the muscles of the extremities and that the disturbance of the motor apparatus and speech was due to the lesions in the superior cerebellar tract at the site of the red nucleus.

2. *Infantile Tremors and Congenital Nystagmus.*—This article is an attempt to make a classification of the diverse forms in which these affections present themselves. The writers base their work almost entirely on the clinical aspect of the cases and await confirmation of their ideas from the pathological discoveries of the future. They regard the tremors and nystagmus as expressions of an organically diseased central nervous system and base their argument upon the fact that in most instances either hereditary taint or some evidences of anatomical defect, such as exaggerated knee-jerks, atrophies, or epileptic manifestations are present. The case of infantile tremor which they describe belongs to the group of cases diagnosed as spasm nutans, and as indicating the presence of some underlying anatomical lesion, or at least a susceptible nervous system; they call attention to the fact that both the maternal grandfather and uncle were born blind, and that one younger brother had epileptiform attacks.

Congenital nystagmus they classify as follows: (1) Essential nystagmus, the manifestation being an isolated symptom; (2) essential nystagmus with various additional nervous symptoms, such as facial asymmetry, inequality of pupils; (3) essential nystagmus with special nervous symptoms, as exaggerated reflexes and epileptic manifestations; (4) the occurrence of the symptom in the family, either alone or associated with other nervous signs.

The pathological changes, they think, may be found at the root of these affections, the tremors and the nystagmus are malformations of motor cells in the cortex, alterations in the cells of the nuclei of motor nerves of the eye, and faults in the conduction of the axis cylinders due perhaps to changes in the myelin sheath. In certain cases, the writers think the cause will be found to lie in punctate hemorrhages at the time of birth.

3. *A Case of Epileptic Delirium.*—Petit reports this case as showing that the epileptic delirium is composed of two factors, the cerebral state and the epileptic impulse. The first of these exists independent of the patient's epilepsy and consists of the ensemble of his moral nature, his habits and his intellectual attainments. The epileptic impulse provokes the unconscious acts of the delirious state, functionates as an electric

spark, and releases the uncontrollable mental processes. But the unconscious acts of the delirium always have their origin in, and are regulated by, the previous "cerebral state" of the individual. The case cited shows this relationship. The patient, while in his normal condition, was taken to a hospital and later to an asylum for observation, because a few days previously he had been suffering from maniacal delirium associated with complete unconsciousness. Six years later he again becomes maniacal, goes to the police and insists upon being taken to the hospital where he had previously recovered from this state. He had no memory of his doings during that time.

4. *The Hysteria of Saint-Theresa.*—This is a historical study of the mental alienation of one who figured prominently in the ecclesiastical history of the sixteenth century. Rouby gives an account of three cases of suspended animation which have occurred in his practice and he demonstrates the hysterical nature of these attacks.

I. Strauss (New York.)..

MISCELLANY.

Carcinosis of the Central Nervous System. E. Siffert (Münch. med. Woch., May 20, 1902).

The course of carcinosis of the central nervous system is usually rapid, according to this author, and more often involves the cord than the brain. It is of comparatively rare occurrence and generally of a metastatic nature. The metastases vary widely in number and are usually situated at the border between the white and gray matter, not often larger than a cherry, and remain quite distinct from the tissues around them although it is quite possible for them to be completely overlooked at autopsy. There are no symptoms of pressure, for the growth is a gradual substitution. With the invasion of the pia the subarachnoid spaces will soon be affected and because there is less resistance offered here there is always increased growth. In the vertebral canal the tissue takes the shape of an irregular cylindrical tube, developing most fully in the dorsal region, noticeably at the point where the posterior nerve roots have their departure. The predominant pathological lesion may be inflammation and hemorrhage, which are always present. The course is very rapid although it may last for a number of weeks, the symptoms being partly mechanical and partly toxic and the last stage is that of an erosion of the nerve-substances. Unless the primary tumor, usually found in the lung, is apparent, the diagnosis is apt to be somewhat hard, but characteristics such as advanced age, increasing apathy, cachexia developing rapidly, delirium alternating with lucid intervals, speech disturbance somewhat similar to that shown in paresis, absence of choked disk, pain and stiffness of the neck, pain and disturbance of the bladder, should prevent error. Jelliffe.

Reflex Spasm Caused by Ascaris. J. P. Naab (Münch. med. Woch., May 13, 1902).

This author notes that cases of ascaris are often taken for disorders much more serious, as when a child is seized with convulsions, followed by leg coma, and a provisional diagnosis of meningitis is often made, until the stools reveal the real cause of the trouble and the administration of santonin and calomel quickly rights it. As in some quite severe cases only a few worms have been discovered it would seem to indicate that the number does not govern the severity of the symptom. An aid to diagnosis may be noted in the excessive secretion of saliva.

Jelliffe.

Focal Facial Epilepsy. McCarthy and Francine (Phil. Med. Jour., June 14, 1902).

A case of focal epilepsy, affecting the right side of the face and tongue, followed by a paralysis of these parts which endured from three to five minutes, is reported by these observers. This case, which was that of an old woman, was interesting on account of the paralysis which followed a localized convulsion. The first attack, which was possibly hysterical, followed an accidental wound in the scalp made by a hatpin, but considering the differential diagnosis of an organic lesion from hysteria, it was believed that reflex epilepsy might have been caused by intense pain. On account of the unique distribution of the paralysis in this case the authors believe that there is a small lesion of gradual development localized in the left motor area in or near the face center, which, while not sufficiently developed to cause serious disturbance of the function of the motor cells for the face and tongue, yet by irritation may lead to paroxysmal convulsions followed by paralysis.

W. B. Noyes.

Toe Reflex. H. Levi (Münch. med. Woch., May 27, 1902).

According to this author the Babinski toe-reflex, which is a dorsal flexion of the big toe after irritation of the plantar surface of the foot, is not entirely reliable as an indication of a lesion in the pyramidal tract. The reaction was positive in six per cent. of the cases where disease of the pyramidal tract could be definitely excluded, and only eighty-six per cent. gave a distinct primary plantar flexion. A plantar flexion is always present in functional diseases, but only in a certain number was a Babinski present, and in paralysis agitans, Huntington's chorea and epilepsy it never occurred. This phenomenon was present in two cases of recent hemiplegia, disappearing in a few days, while the paralysis gave place to a paresis, not of a functional nature, however. As no Babinski could be obtained in many cases one would be led to suppose that increased knee-jerks do not invariably signify pyramidal destruction, but on the other hand quite a number of cases are on record where there was a Babinski and without knee-jerks. Jelliffe.

Rest in Bed in Epileptic Delirium. E. Marandon (La Sem. Méd. 1902, No. 21).

This author reports nineteen cases of epilepsy treated by rest in bed during delirium. Of these only one could be said to have received distinct advantage from this treatment. This patient's condition was so improved that the crises which before had lasted five days, when there were hallucinations of sight and hearing, refusal of food, complete sleeplessness, etc., were reduced to only three days and were much less severe. The third seizure after the use of this treatment was one of purely motor elements. The appetite of the patient was good and insomnia was not present. There was no further evidence of the crises of delirium. In three other cases the treatment did not show such good results, the crisis lasting longer, although it was perhaps less severe, but there was not constant relief. In ten cases the method proved absolutely useless and in four it was distinctly harmful. From his obervation of these patients this author has adopted a varying course in the treatment of such cases. After the first active crisis, during which he observes the duration, intensity and general symptoms, if he does not think rest in bed will be conducive of benefit to the patient he does not hesitate to refuse to prescribe it, recommending instead isolation and quiet, which are generally better suited to the desires of the patient than if required to remain in bed. Jelliffe.

Book Reviews

Nervous and Mental Diseases. By Archibald Church, M.D., and Frederick Peterson, M.D. Third edition. W. B. Sanders & Co., Philadelphia and London.

This text-book having passed through two editions in as many years, is now a third time presented to the profession. In this the subject matter has been rearranged and revised with many additions. Here the allied subjects of neurology and insanity, usually treated separately, are wedded and appear under one cover. The work consists of eight hundred pages, of which the last two hundred are devoted to insanity. This union of the two subjects and the very practical spirit of the whole, renders it particularly valuable to the student and practitioner. In the section on nervous diseases, by Dr. Archibald Church, of Chicago, the clinical pictures are clearly drawn with unusual attention to their treatment and management. Pathological anatomy receives less consideration: but the essentials, necessary for a clear conception of the disease, are given.

The illustrations, diagrams, syndrome tabulations and diagnostic tables are admirably selected and adapted.

In the articles on pain, central and spinal localization and the tropho-neuroses are embodied the results of the most recent investigations on these subjects.

The section on mental diseases is by Dr. Frederick Peterson, of New York. The subject of insanity first receives treatment in a very comprehensive but concise manner from the standpoint of etiology, symptomatology, prognosis, treatment and classification. Then follow detailed descriptions of the several types of mental disease, the whole presenting this abstruse subject in a very clear, vivid and interesting manner. J. Ramsay Hunt (New York).

La Démence Précoce. Par le Dr. G. Deny, Médecin de la Salpêtrière et P. Roy, Interne des Hopitaux de Paris.

Ever since Kraepelin in 1893 made use of the term dementia præcox, to characterize a group of fairly well recognized early dementias, this type of disease has received increasing attention. For in the young adult there is no psychosis that is of as much importance both by reason of its frequency and its gravity, as this.

The authors have here given a singularly lucid and telling account of this affection, adopting Kraepelin's modern views of the condition.

Amid the polymorphic modifications of the disease three forms stand out with greater distinctness than the rest: hebephrenia, catatonia and paranoid types.

These main symptom syndromes are excellently summarized in this small volume and it is well worthy of a wide reading.

News and Notes

A NEW BUILDING is soon to be erected at the Delaware State Hospital for the Insane at Farnhurst for the care and treatment of the tuberculous insane. It is to cost $10,000.

THE NECESSITY of passing a new law in Mississippi is being agitated which shall specifically prohibit the sale of cocaine to negroes, on account of the increase of insanity and suicides among the race due to this drug habituation.

CHICAGO is soon to have an institution which shall give special instruction to children with speech disorders, especially to those who have lost this faculty from cerebral disease.

THE PROTESTANT HOSPITAL FOR THE INSANE at Verdun, Quebec, has just opened a new wing for the reception of eighty additional patients. This increase will bring the total population of the hospital to five hundred.

DR. PEARCE BAILEY has been appointed consulting neurologist at the Roosevelt Hospital, New York City.

AT THE LAST SESSION of the state convention of the directors of the poor at Somerset, Pa., a resolution was passed urging the state legislature to purchase Highland Inn and one hundred acres additional, for the purpose of establishing a state hospital for epileptics.

THE NATIONAL ASSOCIATION FOR THE STUDY OF EPILEPSY AND THE CARE AND TREATMENT OF EPILEPSY, had its second annual meeting at the Academy of Medicine, New York, November 5. The following papers were presented: Presidential Address by Dr. Frederick Peterson; "Medical Treatment of Epilepsy," Dr. A. Jacobi; "The Surgical Treatment of Epilepsy," Dr. Roswell Park; "The Pathology of Epilepsy," Dr. A. Meyer; "The Problem of Epilepsy; Some Suggestions for Its Solution, with Demonstration of the Lesion," Dr. L. Pierce Clark, and Dr. Thomas P. Prout; "The Social and Legal Aspect of the Epileptic," Dr. E. J. Spratling. Dr. W. N. Bullard and Dr. William P. Spratling gave addresses. The meeting was largely attended. Dr. Wharton Sinkler was elected president for the ensuing year.

DR. T. D. CROTHERS, of Hartford Co., has been appointed consulting neurologist by the Board of Trustees of the New York Metropolitan Hospital and Dispensary.

DR. EUGENE G. CARPENTER, Superintendent of the Ohio State Hospital at Columbus, died of apoplexy October 19, at the age of forty-four. He was a graduate of the College of Physicians and Surgeons of Baltimore. He was Assistant Superintendent at the Cleveland State Hospital before his transfer to Columbus four years ago.

THE NEW ORLEANS STATE BOARD OF HEALTH reports that the suicide rate among negroes during the past ten years was 8.2 per 100,000, and is increasing rapidly, two-thirds of the increase being in the past ten years. The causes are reported to be due to illness or fear of death, and in some cases a fear of being lynched. The morphine and cocaine habits also figure prominently among the incentives. Suicide is much more common among those of mixed blood, and especially those of mixed French blood.

Resolutions to the following effect were recently passed at the International Conference on the Treatment of the Insane which was held at Antwerp: (1) Confinement for insane be abolished except in dangerous cases (2) The boarding-out system be instituted wherever possible. (3) Schools for the mentally weak be established under medical supervision. (4) Placing patients in asylums be under the entire control of physicians. (5) Forcible restraint in the care of the insane be entirely abolished.

The New York State Commission in Lunacy has recently issued a statement declaring without foundation in fact, the assertions recently made by the political opponents of the Governor, regarding the State Hospitals for the Insane. These assertions were to the effect that owing to decreased appropriation the efficiency of the State Hospitals for the Insane had been seriously impaired; that scurvy has resulted from diminished food supply and that the hospital officials had in various ways been intimidated. The Commission defends the changes made in the pathological department and says that in consequence of improved methods of research introduced by Dr. Meyer, there is reason to hope that at no distant day tangible results will be manifest in an increase of the recovery rate. It is admitted that the hospitals are overcrowded, but it is asserted that if the contractors having buildings now in course of construction, adequate appropriation for which has been made, had complied with the terms of the contract, requiring completion of buildings for the present year, such accommodation would have been sufficient to have met every requirement. No case of scurvy the Commission states has occurred in any State Hospital since the inauguration of the State Care Act, as far as can be learned by those in responsible authority of the State Hospital. It can be asserted furthermore that at this time and for several years past the insane of the State are and have been receiving better quantity, quality and variety of food than any similar class of persons is receiving anywhere else in the United States. Regarding intimidation charges, the reports say that the rules and regulations which have been put in operation by the Commission and approved by the Governor, were prepared by the Medical Superintendents themselves, the very slight modification therein having been made by the Medical Commissioner, Dr. Frederick Peterson.

The Beverly Farm, Home and School for Feeble-Minded Children at Godfrey, Ill., has recently enlarged its equipment at an expense of $6,000, introducing a kindergarten school and an exclusive cottage for boys.

Trustees of the Illinois Western Hospital for the Insane at Watertown, petitioned the legislature for an appropriation of $450,000, the chief item of which was a new ward building at an estimated cost of $200,000.

A new journal upon Neurology, Psychiatry, Psychology and allied sciences, has recently been established in Tokio, Japan, by Professor Dr. S. Kure and Dr. K. Miura.

The Central Indiana State Hospital opened a new building on November 5 for the sick insane. The cost of the new structure was $132,000.

Do our readers realize in getting the Journal of Nervous and Mental Disease they have abstracts of *all*—not only one or two articles—from the following journals?

English—*The American Journal of Insanity, Brain, The Journal of Mental Science, The Alienist and Neurologist.*

German—*Allgemeine Zeitschrift f. Psychiatrie, Archiv f. Psychiatrie und Nervenkrankheiten, Beitrage zur Psychiatrischen Klinik, Centralblatt f. Nervenheilkunde und Psychiatrie, Deutsche Zeitschrift f. Nervenheilkunde, Jahrbücher f. Psychiatrie und Neurologie, Monatsschrift f. Psychiatrie und Neurologie, Neurologisches Centralblatt, Archiv f. Kriminal Anthropologie.*

French—*Annales Medico-Psychologiques, Archives d'Electricité Medicale, Archives de Neurologie, Journal de Neurologie, Nouvelle Iconographie de la Salpêtrière, Revue Neurologique.*

Italian—*Archivio di Psichiatria Scienze Penali ed Anthropologia Criminale, Rivista di Patologia Nervosa e Mentale, Rivista Sperimentale di Freniatria, Annali di Nevrologia.*

Norwegian—*Tidsskrift for Nordisk Retsmedicin og Psykiatri.*

In addition the general literature of the entire world is carefully scanned and abstracted for our readers.

A Word from the Business Manager.

In closing the year, the first of the new management, the Managing Editor desires to express his thanks for the aid and many helpful suggestions of the readers of the Journal. It is largely due to the generous support of the Editorial Board and to the subscribers that the Journal has had such a successful year.

We desire to thank particularly the directors and superintendents of the sanitaria who have stood by the Journal in greater numbers than ever before, and we take this occasion to remind our readers that nowhere can there be found such a valuable collection of institutions as is represented in the Directory of the Journal of Nervous and Mental Disease. We trust that the co-operation of the sanitaria will meet with the returns from our readers which they so richly deserve.

A word of thanks is also due and cheerfully rendered to the many advertisers who have contributed so widely to the success of the year's undertaking. There is no special journal in the United States that receives better support from the manufacturers of pharmaceuticals than ours, and we trust that our well wishers will remember this, and further that they will bear in mind the many warnings concerning substitution that have appeared in the Publishers' Announcements from time to time. The suit won by the Breitenbach Company concerning infringements of their Gude's Pepto-Mangan, is an instance of a righteous judgment on all such frauds. Similar attempts are being made in Massachusetts to substitute inferior products for Phillip's Milk of Magnesia and the products of Fougera, particularly their Colchi-sal, are being extensively substituted. We bespeak for these and other frauds a watchful eye on the part of our subscribers, who are desirous of getting a good article. We further desire to ask for a hearty endorsement of the generous support of the advertisers who have been so largely instrumental in aiding a journal of such a special character to exist.

For the coming year we hope to give a better journal than ever before, and we trust that the generous support given the management during 1902 will be continued during the years to come. Respectfully,

Smith Ely Jelliffe, M.D., Managing Editor.

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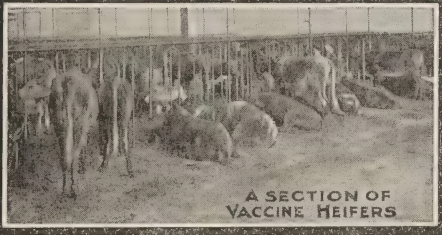

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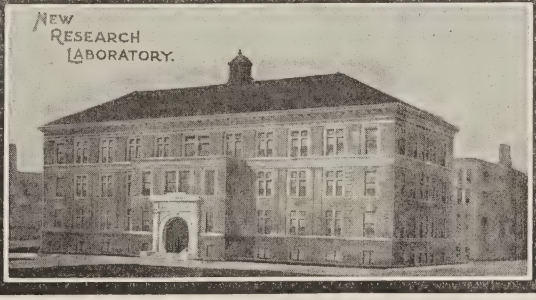

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Ergoapiol (Smith) in Diseases of the Female.

By Charles H. Shepard, M.D., Physician to Lincoln Hospital, Durham, N. C.

A deep and general interest is attached to all knowledge pertaining to the treatment of common diseases of the uterus, to which women are subject, and a vast literature is the outcome of this profound and focused interest. We live today in an age of transition—a period of change. A great many of the former theories in medicine are fast passing away. New medicines are made, achieve a short-lived success, and then pass on to obscurity. This is true most especially in medicines for gynecological diseases. Of the newer remedies it is hard indeed to get one that may be depended upon for long. They soon lose their reputation and potency and are relegated to the past.

We know that all diseases of the womb have not the same etiology nor the same pathology, therefore they should not all have the same treatment. Far too often the general practitioner groups all these diseases together as one and gives the routine treatment. It is not enough to give anodyne medicines for dysmenorrhea no more than it is sufficient to treat alike all forms of dysmenorrhea.

The operation of curettement has a most important place in these conditions, but like other remedial agencies it has its limitation. When we curette the uterus we rid it of a pathologically obnoxious lining membrane, and afford a normal membrane the opportunity to be formed.

The healthy woman with normal genitalia menstruates regularly and painlessly once a month from puberty to the "turn" of life, except that this regularity is interrupted by pregnancy and afterwards by lactation. Any departure from this rule constitutes an abnormality. Amenorrhea is less frequently met with than dysmenorrhea and irregular menstruation. The present age of transition has brought forth what is popularly known as the "new woman," and she has brought with her new ideas and practices which in very many cases retard growth and the natural process necessary for perfect health. For leaving the old landmarks she has to suffer.

The most generally useful medicine in the conditions of amenorrhea, dysmenorrhea, irregular, scanty and fetid menstruation, in my judgment, is a preparation of the Martin H. Smith Company of New York, known as Ergoapiol (Smith). In the female ward of the Lincoln Hospital, Durham, N. C., I have used this medicine very extensively, and it has not only never failed to benefit and cure, but I know no remedy with which I could replace it were I deprived of it. Its efficacy may be tested by any physician who properly tries it. I mention a few cases with short description of each, in which it has given the most signal benefit in my hands.

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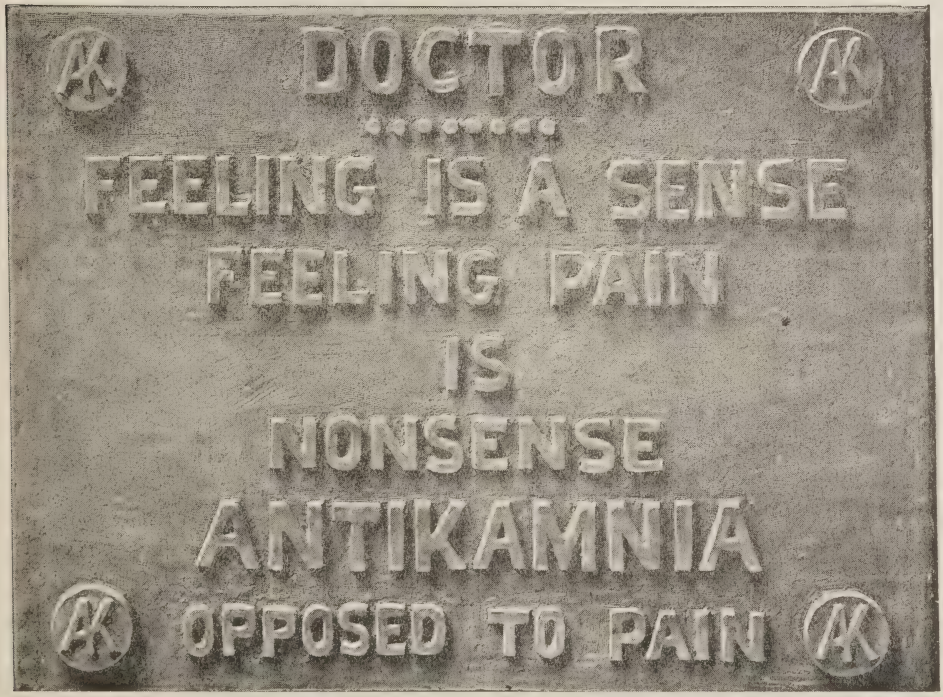

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ing of malaise. The following morning she found to her delightful surprise that she was menstruating for the first time in seven years. At that time the flow was somewhat scanty, but the treatment was continued through three periods. Each succeeding period was more nearly normal than the one that preceded it. Now her functions are regular and I know no reason why she may not become pregnant.

Case No. 2—Mrs. S. complained of a continuous, dull, dragging pain, situated in the region of the iliac fossa of the right side. Menstruation irregular, scanty, fetid. Married six years; had never been pregnant. Excessive leucorrhea, though otherwise she was perfectly normal. Her weight was 140 pounds. Her condition and the suffering, both physical and mental, which it occasioned her, was rapidly undermining her health. She was becoming emaciated, appetite of no consequence, general weakness. She considered her condition "hopeless." Cardiac weakness, of which she was a victim, contra-indicated curettement—which usually cures "whites" and allows the formation of a healthy lining membrane. Ergoapiol (Smith) was prescribed for her, one capsule three times a day. In conjunction with this I gave tonic medicines. After six weeks' use of this remedy the woman said she was "feeling so good" that she did not need any further treatment. She had increased in weight and her appetite had become all she could wish. The menstrual flow was increased, and now, five periods having elapsed from the time treatment was instituted, her monthly flow has failed to appear. She does not expect its return for some time—supposing herself pregnant.

No. 3—Miss S. suffered severe pain each month beginning a day before the flow came on. The flow was a thick, clotted mass, consisting of membrane and the menstrual blood matted together. She had suffered from puberty and the suffering became more intense as the years passed on. She was 19 years of age, stout, of healthy parentage. Admitted to Lincoln Hospital January 15, 1902. She declined an operation. I afterwards prescribed Ergoapiol (Smith) and have continued it for one month. Her next menstruation was free and easy; painless and regular. I doubt not that keeping up this treatment up to another period she will be entirely rid of the hitherto troublesome condition.

No. 4—Miss W. Tubercular history. Menstruation very irregular, sometimes three, sometimes five weeks between periods; very painful; scanty. I prescribed Ergoapiol (Smith) one capsule four times a day beginning one week before the menstrual period and continued a week after the period. As a result of this treatment the patient feels a great deal better in her general health; her monthly flow has been rendered painless and increased in quantity. Ergoapiol has a tonic action upon the muscular fibers of the womb. Its effect is not transitory, but lasting. This superior preparation is decidedly tonic.

No. 5—Mrs. D. A victim of endometritis. Pain continues between periods and is aggravated at periods. Leucorrhea was very pronounced; pains in the back; "hot flushes"; vertigo; headache. Patient would not allow an operation; highly sensitive. Several preparations were tried, but none gave relief until Ergoapiol (Smith) was used. It has entirely relieved the patient and she is now loudly singing its praises. In this case treatment was kept up for ten weeks.

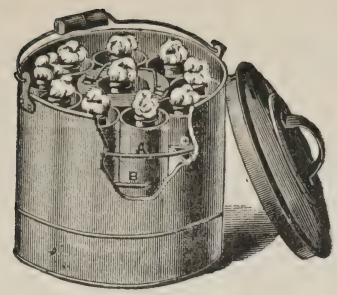
Ergoapiol has never failed in my hands. It is not possible that it can cure obstructive dysmenorrhea, but with that exception it is indicated in all the other diseases of the womb where a tonic and sedative action is the requirement.

Case No. 6—Mrs. D., widow; aged 33; had three children; youngest ten years of age. She had suffered all her menstrual life severe pains in the pelvis at each period; had to keep in bed a week or more each month; paroxysms of pain were followed by a flow of the "whites"; no anemia; womb found to be flabby and relaxed; pains extended down thighs posteriorly. Had been treated for many years by various physicians of note, but had received only temporary benefit.

Ergoapiol (Smith) was given her, one capsule three times a day and increased at the time of the flow to four a day. After three months of this treatment her menstrual function became regular, and being entirely well now she feels that life after all is worth living.

I could prolong this list indefinitely with records of cases that have been entirely relieved of these conditions, and I shall be pleased to furnish any information as to Ergoapiol (Smith) and its use.

No physician can afford to be indifferent regarding the accurate filling of his prescription.



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